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REGULATORY DETERMINATIONS SUPPORT DOCUMENT FOR SELECTED CONTAMINANTS FROM THE SECOND DRINKING WATER CONTAMINANT CANDIDATE LIST (CCL 2)

Disclaimer

This document is designed to provide technical background information for the regulatory determinations being considered by the Office of Ground Water and Drinking Water.

This document is not a regulation itself, and it does not substitute for the Safe Drinking Water Act (SDWA) or the Environmental Protection Agency's (EPA's) regulations. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Acknowledgments

The compilation and analysis of information and data presented in this report were undertaken by EPA's Office of Ground Water and Drinking Water (OGWDW) in support of their determinations regarding whether regulating specific CCL 2 drinking water contaminants will present a meaningful opportunity to reduce health risk. This effort was directed by Mr. Clifton Townsend and Ms. Wynne Miller of OGWDW's Standards and Risk Management Division (SRMD) Targeting and Analysis Branch (TAB). Wynne Miller served as the Team Lead for the CCL 2 Preliminary Regulatory Determinations under the guidance of Ann Codrington (TAB Associate Branch Chief until December 2005), Eric Burneson (TAB Branch Chief), Phil Oshida (SRMD Deputy Division Director), Pamela Barr (SRMD Division Director) and Cynthia Dougherty (OGWDW Office Director).

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Executive Summary

This document provides background information to support EPA's preliminary regulatory determinations for drinking water contaminants on the second Contaminant Candidate List (CCL 2). The preliminary regulatory determinations are presented formally in the *Federal Register*. This report itself does not constitute regulation.

This regulatory support document is divided into three Parts and fifteen Chapters. Because EPA understands that members of the public with varied concerns might be more interested in certain contaminants and less interested in others, the document is designed in such a way that individual chapters are more or less self-contained and can be distributed separately.

Part I, which includes the first two Chapters, provides preliminary information. Chapter 1 is an introduction to the CCL and regulatory determination process. Chapter 2 provides general information on the most important sources of data used to evaluate contaminants.

Chapters 3 through 11, in Part II, discuss eleven of the 51 CCL 2 contaminants for which EPA is making a preliminary regulatory determination. These contaminants are: boron, dimethyl tetrachloroterephthalate (DCPA) mono- and di-acid degradates, DDE, 1,3-dichloropropene, 2,4- and 2,6-dinitrotoluene, EPTC, fonofos, terbacil, and 1,1,2,2,-tetrachloroethane. Each chapter includes information on contaminant properties and sources, environmental fate and behavior, health effects, use and environmental release, known occurrence in ambient water and drinking water, and available analytical methods and treatment technologies. For each of these contaminants, EPA has made a preliminary determination that in light of available data, a national primary drinking water regulation (NPDWR) is not warranted. Those decisions are presented formally in the *Federal Register*. In some cases, EPA intends to update existing Health Advisories and/or provide guidance to states that face local contamination problems.

EPA has not made preliminary regulatory determinations for the remaining CCL 2 contaminants. Because EPA understands that members of the public may have a particular interest in certain high-profile CCL 2 contaminants, Chapters 12 through 15, in Part III, discuss the status of EPA's evaluation of perchlorate, metolachlor, MTBE, and several microbiological contaminants. EPA is not precluded from making regulatory determinations on any of these contaminants before the next round of formal CCL regulatory determinations.

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A chapter from:

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Abbreviations

ATSDR	Agency for Toxic Substances and Disease Registry
CCL	Contaminant Candidate List
CCL 1	First Contaminant Candidate List
CCL 2	Second Contaminant Candidate List
DDE	1,1-Dichloro-2,2-bis(*p*-chlorophenyl) ethylene
EPTC	s-Ethyl dipropylthiocarbamate
HRL	Health Reference Level
IRIS	Integrated Risk Information System
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MTBE	Methyl tertiary-butyl ether
NAS	National Academy of Sciences
NDWAC	National Drinking Water Advisory Council
NPDWR	National Primary Drinking Water Regulation
NRC	National Research Council
OPP	Office of Pesticide Programs
PWS	Public Water System
RED	Reregistration Eligibility Decisions
SAB	Science Advisory Board
SDWA	Safe Drinking Water Act

1 Introduction

1.1 Purpose and Scope

The 1996 Safe Drinking Water Act (SDWA) Amendments (section 1412(b)(1)) direct EPA to publish a list of currently unregulated contaminants that may pose risks for drinking water (referred to as the Contaminant Candidate List, or CCL) and to make determinations on whether to regulate at least five contaminants from the CCL with a national primary drinking water regulation (NPDWR). This regulatory determination support document provides:

(1) a summary of the statutory requirements and previous activities related to the contaminant candidate list and regulatory determinations,

(2) the approach used to identify and evaluate contaminants for the Agency's second round of regulatory determinations,

(3) information and data on the physical and chemical properties, use and environmental release, environmental fate, potential health effects, and occurrence and exposure estimates for each of the 11 contaminants that the Agency evaluated,

(4) the preliminary determination for each of the 11 contaminant candidates, and

(5) the Agency's rationale for its regulatory determination for these 11 contaminants

The 11 regulatory determination candidates discussed in this document include boron, the dacthal mono- and di-acid degradates, 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE), 1,3-dichloropropene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, s-ethyl dipropylthiocarbamate (EPTC), fonofos, terbacil, and 1,1,2,2-tetrachloroethane.

Additionally, this support document includes information and data on several contaminants for which no regulatory determination has been made at this time. These include perchlorate, metolachlor, methyl tertiary-butyl ether (MTBE), and nine microbial contaminants.

1.2 Background on the CCL and Regulatory Determinations

1.2.1 Statutory Requirements for CCL and Regulatory Determinations

The specific statutory requirements for the CCL and regulatory determinations can be found in SDWA Section 1412(b)(1). The 1996 SDWA Amendments require EPA to publish the CCL every five years. The CCL is a list of contaminants that are not subject to any proposed or promulgated NPDWRs, are known or anticipated to occur in public water systems (PWSs), and may require regulation under SDWA. The 1996 SDWA Amendments also direct EPA to determine whether to regulate at least five contaminants from the CCL every five years (within three and one-half years after publication of the final list). In making regulatory determinations,

SDWA requires EPA to publish a Maximum Contaminant Level Goal[1] (MCLG) and promulgate an NPDWR[2] for a contaminant if the Administrator determines that:

(a) the contaminant may have an adverse effect on the health of persons;
(b) the contaminant is known to occur or there is substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern; and
(c) in the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems.

If EPA determines that all three of these statutory criteria are met and makes a final determination that an NPDWR is needed, the Agency has 24 months to publish a proposed MCLG and NPDWR. After the proposal, the Agency has 18 months to publish and promulgate a final MCLG and NPDWR (SDWA section 1412(b)(1)(E)).[3]

1.2.2 The First Contaminant Candidate List (CCL 1)

Following the 1996 SDWA Amendments, EPA sought input from the National Drinking Water Advisory Council (NDWAC) on the process that should be used to identify contaminants for inclusion on the CCL. For chemical contaminants, the Agency developed screening and evaluation criteria based on recommendations from NDWAC. For microbiological contaminants, NDWAC recommended that the Agency seek external expertise to identify and select potential waterborne pathogens. As a result, the Agency convened a workshop of microbiologists and public health experts who developed criteria for screening and evaluation and subsequently developed an initial list of potential microbiological contaminants.

The first CCL process benefited from considerable input from the NDWAC, the scientific community, and the public through stakeholder meetings and the public comments received on the draft CCL published on October 6, 1997 (62 FR 52193). EPA published the final CCL, which contained 50 chemical and 10 microbiological contaminants, on March 2, 1998 (63 FR 10273). A more detailed discussion of how EPA developed CCL 1 can be found in the 1997 and the 1998 *Federal Register* notices (62 FR 52193 and 63 FR 10273).

1.2.3 The Regulatory Determinations for CCL 1

EPA published its preliminary regulatory determinations for a subset of contaminants listed on CCL 1 on June 3, 2002 (67 FR 38222). The Agency published its final regulatory

[1] The MCLG is the "maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur, and which allows an adequate margin of safety. Maximum contaminant level goals are nonenforceable health goals." (CFR 141.2)

[2] An NPDWR is a legally enforceable standard that applies to public water systems. An NPDWR sets a legal limit (called a maximum contaminant level or MCL) or specifies a certain treatment technique (TT) for public water systems for a specific contaminant or group of contaminants.

[3] The statute authorizes a nine month extension of this promulgation date.

determinations on July 18, 2003 (68 FR 42898). EPA identified 9 contaminants from the 60 contaminants listed on CCL 1 that had sufficient data and information available to make regulatory determinations. The nine contaminants were *Acanthamoeba*, aldrin, dieldrin, hexachlorobutadiene, manganese, metribuzin, naphthalene, sodium, and sulfate. The Agency determined that an NPDWR was not necessary for any of these nine contaminants. The Agency issued guidance on *Acanthamoeba* and health advisories for magnesium, sodium, and sulfate.

The decision-making process that EPA used to make its regulatory determinations for CCL 1 was based on substantial expert input and recommendations from different groups including stakeholders, the National Research Council (NRC), and NDWAC. In June 2002, EPA consulted with the Science Advisory Board (SAB) Drinking Water Committee and requested its review and comment on whether the protocol EPA developed, based on the NDWAC recommendations, was consistently applied and appropriately documented. SAB provided verbal feedback regarding the use of the NRC and NDWAC recommendations in EPA's decision criteria for making its regulatory determinations. SAB recommended that the Agency provide a transparent and clear explanation of the process for making regulatory determinations. The Agency took SAB's recommendation into consideration and further explained the CCL 1 regulatory determination evaluation process in the July 18, 2003 (68 FR 42898) notice and in the supporting documentation.

EPA has used the same approach for the present round of regulatory determinations. While this document includes a short description of the decision process used to make regulatory determinations (see section 1.3, below), a more detailed discussion can be found in the 2002 and the 2003 *Federal Register* notices (67 FR 38222 and 68 FR 42898).

1.2.4 The Second Contaminant Candidate List (CCL 2)

The Agency published its draft CCL 2 *Federal Register* notice on April 2, 2004 (69 FR 17406) and the final CCL 2 *Federal Register* notice on February 24, 2005 (70 FR 9071). The CCL 2 carried forward the 51 remaining chemical and microbial contaminants that were listed on CCL 1.

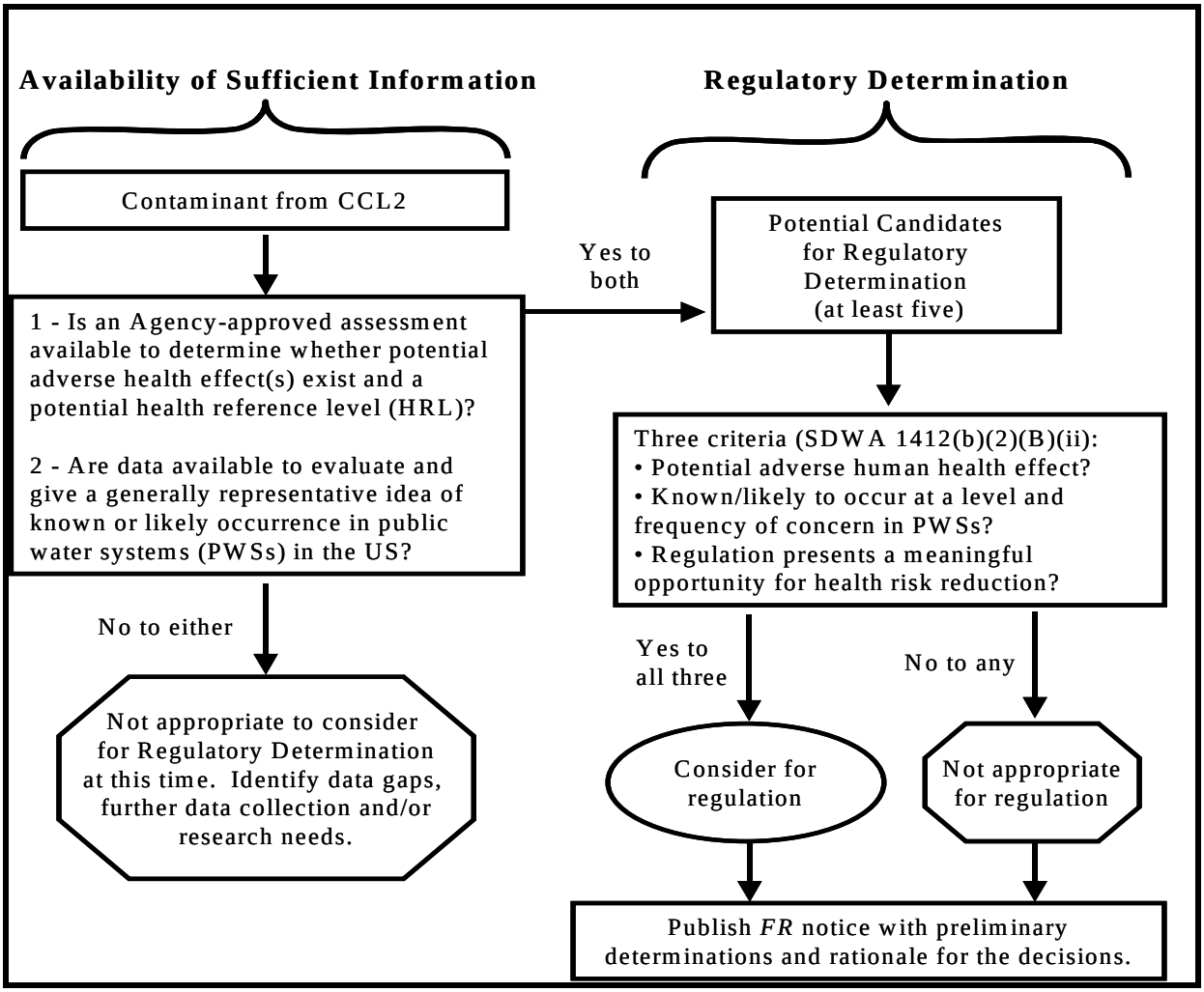
1.2.5 The Regulatory Determinations for CCL 2

As discussed below, EPA has made preliminary determinations for 11 of the 51 contaminants listed on the CCL 2.

1.3 Summary of the Approach Used to Identify and Evaluate Candidates for Regulatory Determination 2

Exhibit 1-1 provides a brief overview of the process EPA used to identify which CCL 2 contaminants are candidates for regulatory determinations and the SDWA statutory criteria considered in making the regulatory determinations.

Exhibit 1-1: General Overview of the Approach Used to Evaluate CCL 2 Contaminants for Regulatory Determinations



In identifying which CCL 2 contaminants are candidates for regulatory determinations, the Agency considered whether sufficient information and/or data were available to characterize the potential health effects and the known/likely occurrence in and exposure from drinking water. With regards to sufficient health effects information/data, the Agency considered whether an Agency-approved health risk assessment[4] was available to identify any potential adverse health effect(s) and derive an estimated level at which adverse health effect(s) are likely to occur. With regards to sufficient occurrence information/data, the Agency considered whether information/ data were available to evaluate and give a generally representative idea of known and/or likely occurrence in public water systems. If sufficient information/data were available to

[4] Health information used for the regulatory determinations process includes but is not limited to health assessments available from the Agency's Integrated Risk Information System (IRIS), the Agency's Office of Pesticide Programs (OPP) in a Reregistration Eligibility Decision (RED), the National Academy of Sciences (NAS), and/or the Agency for Toxic Substances and Disease Registry (ATSDR).

characterize adverse human health effects and known/likely occurrence in public water systems, the Agency identified the contaminant as a potential candidate for regulatory determinations. In addition to information/data for health and occurrence, EPA also considered the availability and adequacy of analytical methods (for monitoring) and treatment.

If EPA chose a contaminant as a candidate for regulatory determination, the Agency used an approach similar to the first regulatory determination process to answer the three statutory criteria (listed above, in section 1.2.1).

For the current regulatory determination process, the Agency considered the following in evaluating each of the three statutory criteria.

(1) First statutory criterion - Is the contaminant likely to cause an adverse effect on the health of persons? The Agency evaluated the best available, peer-reviewed assessments and studies to characterize the human health effects that may result from exposure to the contaminant when found in drinking water. Based on this characterization, the Agency estimated a health reference level (HRL) for each contaminant. Section 2.1 provides more detailed information about the approach used to evaluate and analyze the health information.

(2) Second statutory criterion - Is the contaminant known or likely to occur in public water systems at a frequency and level of concern? To evaluate known occurrence in PWSs, the Agency compiled, screened, and analyzed data from several occurrence data sets to develop representative occurrence estimates for public drinking water systems. EPA used the HRL estimates for each contaminant as a benchmark against which to conduct an initial evaluation or screening of the occurrence data. For each contaminant, EPA estimated the number of PWSs (and the population served by these PWSs) with detections greater than one-half the HRL (> ½ HRL) and greater than the HRL (> HRL). To evaluate the likelihood of a contaminant to occur in drinking water, the Agency considered information on use and release into the environment and supplemental information on occurrence in water (e.g. ambient water quality data, State ambient or finished water data, and/or special studies performed by other agencies, organizations, and/or entities). Section 2.2 provides more details on the approach used to analyze the occurrence information/data.

(3) Third statutory criterion - In the sole judgment of the Administrator, does regulation of the contaminant present a meaningful opportunity for health risk reduction for persons served by public water systems? EPA evaluated the potential health effects and the results of the occurrence and exposure estimates (i.e., the population exposed and the sources of exposure) at the health level of concern to determine if regulation presents a meaningful opportunity for health risk reduction. EPA has made a preliminary determination regarding the meaningful opportunity for health risk reduction for eleven contaminants based upon the population exposed to these contaminants at levels of concern.

If the answers to all three statutory criteria are affirmative for a particular contaminant, then the Agency makes a determination that a national drinking water regulation is necessary and

proceeds to develop an MCLG and an NPDWR for that contaminant. It should be noted that this regulatory determination process is independent of the more detailed analyses needed to develop an NPDWR. Thus, a decision to regulate is the beginning of the Agency regulatory development process, not the end.

If the answer to any of the three statutory criteria is negative, then the Agency makes a determination that a national drinking water regulation is not necessary for that contaminant.

1.4 Summary of Preliminary Regulatory Determinations

EPA has made preliminary determinations that no regulatory actions are appropriate for the 11 contaminants evaluated for this second round of regulatory determinations. These 11 contaminants are discussed in detail in Chapters 3 through 11 in Part II of this regulatory determination support document. EPA will make final determinations on these 11 contaminants after a 60-day comment period. EPA is making preliminary regulatory determinations only on those CCL 2 contaminants that have sufficient information to support such a determination at this time. The Agency continues to conduct research and/or to collect information on the remaining CCL 2 contaminants to fill identified data gaps. Some of those contaminants are discussed in Chapters 12 through 15 in Part III of this document. The Agency is not precluded from taking action when information becomes available and will not necessarily wait until the end of the next regulatory determination cycle before making other regulatory determinations.

Chapter 2: Evaluation of Health and Occurrence Data

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

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Abbreviations

AWWARF	American Water Works Association Research Foundation
BW	Body Weight
CCL	Contaminant Candidate List
CCL 2	Second Contaminant Candidate List
CMR	Chemical Monitoring Reform
CWS	Community Water System
CWSS	Community Water System Survey
1,3-DCP	1,3-Dichloropropene
DCPA	Dimethyl tetrachloroterephthalate
DDE	p,p-Dichlorodiphenyldichloroethylene or 1,1-Dichloro-2,2-bis(*p*-chlorophenyl)ethylene
DWI	Drinking Water Intake
EPCRA	Emergency Planning and Community Right-to-Know
EPTC	s-Ethyl dipropylthiocarbamate
HRL	Health Reference Level
IOC	Inorganic Compound
IRIS	Integrated Risk Information System
LOAEL	Lowest Observed Adverse Effect Level
MCLG	Maximum Contaminant Level Goal
MRL	Minimum Reporting Level
MTBE	Methyl tertiary-butyl ether
MTP	Monomethyl tetrachloroterephthalic acid
NAWQA	National Water Quality Assessment
NCFAP	National Center for Food and Agricultural Policy
NCOD	National Drinking Water Contaminant Occurrence Database
NIRS	National Inorganics and Radionuclides Survey
NOAEL	No Observed Adverse Effect Level
NPDES	National Pollutant Discharge Elimination System
NPS	National Pesticide Survey
NURP	Nationwide Urban Runoff Program
OPP	Office of Pesticide Programs
PGWDB	Pesticides in Ground Water Database
PWS	Public Water System
RED	Reregistration Eligibility Decisions
RfD	Reference Dose
RL	Reporting Level
RSC	Relative Source Contribution
SDWA	Safe Drinking Water Act
SOC	Synthetic Organic Compound

SVOC	Semi-volatile Organic Compound
TPA	Tetrachloroterephthalic acid
TRI	Toxics Release Inventory
UCM	Unregulated Contaminant Monitoring
UCMR	Unregulated Contaminant Monitoring Regulation
UCMR 1	First Unregulated Contaminant Monitoring Regulation
UF	Uncertainty Factor
USGS	United States Geological Survey
VOC	Volatile Organic Compound

2 Evaluation of Health and Occurrence Data

2.1 Evaluation of Adverse Health Effects

Section 1412(b)(1)(A)(i) of the Safe Drinking Water Act (SDWA) requires EPA to determine whether each candidate contaminant may have an adverse effect on public health. This section describes the overall process the Agency used to evaluate health effects information, the approach used to estimate a contaminant health reference level or HRL (a benchmark against which to conduct the initial evaluation of the occurrence data), and the approach used to identify and evaluate information on hazard and dose-response for the contaminants under consideration. More specific information about the potential for adverse health effects for each contaminant is included in Part II of this document ("CCL 2 Contaminants Undergoing Regulatory Determination").

There are two different approaches to the derivation of an HRL. One approach is used for chemicals that cause cancer and exhibit a linear response to dose. The other applies to noncarcinogens and carcinogens evaluated using a non-linear approach.

2.1.1 Use of Carcinogenicity Data for the Derivation of a Health Reference Level

For those contaminants considered to be likely or probable human carcinogens, EPA evaluated data on the mode of action of the chemical to determine the method of low dose extrapolation. When this analysis indicates that a linear low dose extrapolation is appropriate or when data on the mode of action are lacking, EPA uses a low dose linear extrapolation to calculate risk-specific doses. The risk-specific doses are the estimated oral exposures associated with lifetime excess risk levels that range from one cancer in ten thousand (10^{-4}) to one cancer in a million (10^{-6}). The risk-specific doses (expressed as mg/kg of body weight per day) are combined with adult body weight and drinking water consumption data to estimate drinking water concentrations corresponding to this risk range. EPA generally used the one-in-a-million (10^{-6}) cancer risk in the initial screening of the occurrence data for carcinogens evaluated using linear low dose extrapolation. Five of the eleven contaminants undergoing regulatory determination had data available to classify them as likely or probable human carcinogens. These five are also the only contaminants for which low dose linear extrapolations were performed. These five are *p,p*-dichlorodiphenyldichloroethylene (DDE), 1,3-dichloropropene (1,3-DCP or Telone), 2,4-dinitrotoluene, 2,6-dinitrotoluene, and 1,1,2,2-tetrachloroethane. The remaining six contaminants have not been identified as known, or likely, or probable carcinogens.

2.1.2 Use of Non-carcinogenic Health Effects Data for Derivation of an HRL

For those chemicals not considered to be carcinogenic to humans, EPA generally calculates a reference dose (RfD). A RfD is an estimate of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from either a "no-observed-adverse-effect level" (NOAEL), a "lowest-observed-adverse-effect level" (LOAEL), or a benchmark dose, with uncertainty factors applied to reflect limitations of the data used.

The Agency uses uncertainty factors (UFs) to address uncertainty resulting from incompleteness of the toxicological database. The individual UFs (usually applied as integers of one, three, or ten) are multiplied together and used to derive the RfD from experimental data. Individual UFs are intended to account for:

(1) the variation in sensitivity among the members of the human population (i.e., intraspecies variability);
(2) the uncertainty in extrapolating animal data to humans (i.e., interspecies variability);
(3) the uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure to lifetime exposure (i.e., extrapolating from subchronic to chronic exposure);
(4) the uncertainty in extrapolating from a LOAEL rather than from a NOAEL; and/or
(5) the uncertainty associated with an incomplete database.

For boron, the dacthal (dimethyl tetrachloroterephthalate or DCPA) mono- and di-acid degradates, s-ethyl dipropylthiocarbamate (EPTC), fonofos, and terbacil, EPA derived the HRLs using the RfD approach as follows:

$$HRL = [(RfD \times BW)/DWI] \times RSC$$

Where:
RfD = Reference Dose
BW = Body Weight for an adult, assumed to be 70 kilograms (kg)
DWI = Drinking Water Intake, assumed to be 2 L/day (90th percentile)
RSC = Relative Source Contribution, or the level of exposure believed to result from drinking water when compared to other sources (e.g., food, ambient air).

A 20 percent RSC is being used to estimate the HRL and screen the occurrence data because it is the lowest and most conservative RSC used in the derivation of a maximum contaminant level goal (MCLG) for drinking water. For each of the six aforementioned non-carcinogenic compounds for which the Agency has made a preliminary regulatory determination, EPA used the RfD in conjunction with a 20 percent RSC to derive a conservative HRL estimate and perform an initial screening of the drinking water occurrence data. Since the initial screening of the occurrence data at this conservative HRL value resulted in a preliminary negative determination for each of these 6 compounds, the Agency determined that it was not necessary to further evaluate the RSC in making the regulatory determination.

As discussed in Chapter 4, the HRL for the two DCPA degradates is based on the HRL value derived for the parent compound following the guidance provided by the EPA's Office of Pesticide Programs.

2.1.3 Sources of Data/Information for Health Effects

EPA used the best available peer-reviewed data and analyses in evaluating adverse health effects. Peer-reviewed health-risk assessments were available for all chemicals considered for regulatory determinations from the Agency's Integrated Risk Information System (IRIS)

Program[1] and/or the Office of Pesticide Programs (OPP) Reregistration Eligibility Decisions (RED)[2]. Exhibit 2-1 summarizes the sources of the health assessment data for each chemical under regulatory determination consideration. The Agency performed a literature search for studies published after the IRIS or OPP health-risk assessment was completed to determine if new information suggested a different outcome. The Agency collected and evaluated any peer-reviewed publications identified through the literature search for their impact on the RfD and/or cancer assessment. In cases where the recent data indicated that a change to the existing RfD or cancer assessment was needed, the updated OW assessment, as described in the health effects support document, was independently peer-reviewed. All quantitative cancer assessments conducted under the Guidelines for Carcinogen Risk Assessment (USEPA, 1986) were updated using the Guidelines for Carcinogen Risk Assessment (USEPA, 1999) as directed in the November 2001 (66 FR 59593) Federal Register notice (USEPA, 2001a).

In March 2005, EPA updated and finalized the Cancer Guidelines and a Supplementary Children's Guidance, which include new considerations for mode of action and added guidelines related to potential risks due to early childhood exposure (USEPA, 2005a; USEPA, 2005b). EPA updated the earlier assessments (based on the 1986 Guidelines) for DDE, the dinitrotoluenes (2,4 and 2,6 as a mixture), and 1,1,2,2-tetrachloroethane following the 1999 Guidelines. None of these chemicals have been determined to have a mutagenic mode of action, which would require an extra factor of safety for children's health protection. Therefore, conducting the cancer evaluation using the 2005 Cancer Guidelines would not result in any change from the assessment updated following the 1999 Guidelines.

The cancer assessment for 1,3-dichloropropene was done by OPP and IRIS (USEPA, 1998 and 2000a) under the Proposed Guidelines for Carcinogen Risk Assessment (61 FR 17960). The Administrator (USEPA, 2005c) has directed that current completed assessments can be considered to be scientifically sound based on the guidance used when the assessment was completed until a new assessment is performed by one of the responsible program offices.

[1] IRIS is an electronic EPA data base (www.epa.gov/iris/index.html) containing peer-reviewed information on human health effects that may result from exposure to various chemicals in the environment. These chemical files contain descriptive and quantitative information on hazard identification and dose response, RfDs for chronic noncarcinogenic health effects, as well as slope factors and unit risks for carcinogenic effects.

[2] The OPP is required under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA) to review all pesticides registered prior to 1984 and determine whether to reregister them for continued use. The results of the reregistration analysis are included in the REDs. Copies of the REDs are located at the following web site: http://cfpub.epa.gov/oppref/rereg/status.cfm?show=rereg.

Exhibit 2-1: Sources and Dates of EPA Health Risk Assessments

Chemical	IRIS	Date	OPP RED	Date
Boron	X	2004	---	---
Dacthal and its mono- and di-acid degradates	X	1994	X	1998
1,3-Dichloropropene	X	2000	X	1998
DDE	X	1988	---	---
2,4-Dinitrotoluene	X	1990/1992	---	---
2,6-Dinitrotoluene	X*	1990	---	---
EPTC	X	1990	X	1999
Fonofos	X	1991	X**	1996
Terbacil	X	1989	X	1998
1,1,2,2-Tetrachloroethane	X	1986	---	---

* Applies to a mixture of 98 percent 2,4-dinitrotoluene and 2 percent 2,6-dinitrotoluene
** Health Risk Assessment; RED not completed due to pesticide cancellation.

EPA has prepared several technical health effects support documents[3] for the contaminants considered for this round of regulatory determinations. These documents address the exposure from drinking water and other media, toxicokinetics, hazard identification, and dose-response assessment, and provide an overall characterization of risk from drinking water.

[3] The health support documents include the following documents: *Health Effects Support Document for Boron* (EPA-822-R-06-005), *Health Effects Support Document for Dacthal Degradates: Tetrachloroterephthalic Acid (TPA) and Monomethyl Tetrachloroterephthalic Acid (MTP)* (EPA-822-R-06-006), *Health Effects Support Document for 1,1-Dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE)* (EPA-822-R-06-007), *Health Effects Support Document for S-Ethyl dipropylthiocarbamate (EPTC)* (EPA-822-R-06-008), *Health Effects Support Document for Fonofos* (EPA-822-R-06-009), *Health Effects Support Document for Terbacil* (EPA-822-R-06-010), *Health Effects Support Document for 1,3-Dichloropropene* (EPA-822-R-06-011), *Health Effects Support Document for 1,1,2,2-Tetrachloroethane* (EPA-822-R-06-012), and *Health Advisory for 2,4- and 2,6-Dinitrotoluene* (EPA-822-R-06-017).

2.2 Evaluation of Contaminant Occurrence and Exposure

EPA used data from several sources to evaluate occurrence and exposure for the 11 contaminants considered in these regulatory determinations. The major or primary sources of the drinking water occurrence data used to support these determinations include the following sources:

- the first Unregulated Contaminant Monitoring Regulation (UCMR 1),
- the Unregulated Contaminant Monitoring (UCM) program, and
- the National Inorganics and Radionuclides Survey (NIRS).

All three are national assessments that were administered or overseen by EPA. General background and methodological information for each of these sources is summarized in Section 2.2.1 below.

In addition to these primary sources of occurrence data, the Agency also evaluated supplemental sources of information on contaminant use and release, occurrence in ambient water, and occurrence in drinking water. These are mostly national assessments by federal agencies such as EPA and the United States Geological Survey (USGS), but they also include regional- and State-level surveys and some research performed by private institutions. Section 2.2.2 provides brief summary descriptions of some of the most important supplemental sources of occurrence information and/or data. A summary of the occurrence data and the results or findings for each of the 11 contaminants considered for regulatory determination is presented in Part II ("CCL 2 Contaminants Undergoing Regulatory Determination").

2.2.1 Primary Data Sources

As previously mentioned, the primary sources of the drinking water occurrence data used to support the regulatory determinations are the UCMR 1, the UCM program, and NIRS. Exhibit 2-2 lists the primary data sources the Agency used for each of the 11 contaminants considered for regulatory determinations.

Exhibit 2-2: Primary Sources of Drinking Water Occurrence Data Used in the Regulatory Determination Process

#	Contaminant	Primary Data Sources				
		UCMR 1		UCM		NIRS
		List 1 Assessment Monitoring	List 2 Screening Survey	Round 1 Cross Section	Round 2 Cross Section	
1	Boron					X[1]
2 3	Dacthal mono- and di-acid degradates	X				
4	DDE	X				
5	1,3-Dichloropropene	X[2]		X	X	
6	2,4-Dinitrotoluene	X				
7	2,6-Dinitrotoluene	X				
8	EPTC	X				
9	Fonofos		X			
10	Terbacil	X				
11	1,1,2,2-Tetrachloroethane			X	X	

1- For boron, EPA also considered the results of a study funded by the American Water Works Association Research Foundation (AWWARF) (Frey *et al.*, 2004).
2- 1,3-Dichloropropene was sampled as a UCM Round 1 and 2 analyte but due to sample degradation concerns the contaminant was re-analyzed using the samples provided by the small systems that participated in the UCMR List 1 Assessment Monitoring.

Occurrence values from the UCMR 1, UCM, and NIRS data sets represent direct counts of the number and percent of systems, and population served by systems, with at least one analytical detection above some specified concentration threshold. EPA considered this to be the most straightforward and accurate way to present these data for the regulatory determination process.

While both UCMR 1 and UCM data could support more involved statistical modeling to characterize occurrence based on mean (rather than peak) concentrations, EPA chose not to perform this step for the regulatory determinations discussed in this document. EPA believes that presenting the actual results of the occurrence monitoring is straight-forward and the use of an analysis based on peak concentrations provides conservative estimates of occurrence and potential exposure from drinking water. Given that the preliminary determinations for the 11 contaminants discussed here are negative, it is not necessary to go beyond the conservative (peak concentration) approach used for this analysis.

The following sections provide a brief summary of the data sources and the approach used to estimate a given contaminant's occurrence. For a more detailed description of the UCM program, see USEPA (2000b) and USEPA (2006a). For a more detailed description of NIRS, please refer to Longtin (1988) and USEPA (2006a). For the UCMR program, please refer to USEPA (2001b) and USEPA (2006b).

The First Unregulated Contaminant Monitoring Regulation (UCMR 1)

In 1999, EPA developed the UCMR program in coordination with the Contaminant Candidate List (CCL) and the National Drinking Water Contaminant Occurrence Database (NCOD) to provide national occurrence information on unregulated contaminants (September 17, 1999, 64 FR 50556; March 2, 2000, 65 FR 11372; and January 11, 2001, 66 FR 2273). EPA used data from the UCMR 1 program to evaluate occurrence for nine of the eleven contaminants considered for these regulatory determinations. These nine contaminants include the dacthal mono- and di-acid degradates, DDE, 1,3-dichloropropene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, EPTC, fonofos, and terbacil.

EPA designed the UCMR 1 data collection with three parts (or tiers), primarily based on the availability of analytical methods. Occurrence data for eight of the nine contaminants listed in the preceding paragraph are from the first tier of UCMR 1 (also known as UCMR 1 List 1 Assessment Monitoring). Occurrence data for fonofos are from the second tier of UCMR 1 (also known as the UCMR 1 List 2 Screening Survey). EPA has not collected data as part of the third tier due to the lack of adequate analytical methods.

The UCMR 1 List 1 Assessment Monitoring was performed for a specified number of chemical contaminants for which analytical methods have been developed. EPA required all large[4] public water systems (PWSs), plus a statistically representative national sample of 800 small[5] PWSs to conduct Assessment Monitoring.[6] Approximately one-third of the participating small systems were scheduled to monitor for these contaminants during each calendar year from 2001 through 2003. Large systems could conduct one year of monitoring anytime during the 2001-2003 UCMR 1 period. EPA specified a quarterly monitoring schedule for surface water systems and a twice-a-year, six-month interval monitoring schedule for ground water systems. The objective of the UCMR sampling approach for small systems was to collect contaminant occurrence data from a statistically selected, nationally representative sample of small systems. The small system sample was stratified and population-weighted, and included some other sampling adjustments such as ensuring the selection of at least two systems from each State. With contaminant monitoring data from all large PWSs and a statistical, nationally representative sample of small PWSs, the UCMR 1 List 1 Assessment Monitoring program provides a contaminant occurrence data set suitable for national drinking water estimates.

In total, 370,312 sample results have been collected under the UCMR 1 List 1 Assessment Monitoring program at 3,083 large systems and 797 small systems. Approximately 33,600 samples were collected for each contaminant. The UCMR 1 List 1 Monitoring program

[4] Systems serving more than 10,000 people.
[5] Systems serving 10,000 people or fewer.
[6] Large and small systems that purchase 100% of their water supply were not required to participate in the UCMR 1 Assessment Monitoring or the UCMR 1 Screening Survey.

included systems from all 50 States, the District of Columbia, four U.S. Territories, and Tribal lands in five EPA Regions. An additional 3,719 samples were collected for 1,3-DCP at all small systems that conducted UCMR 1 List 1 Assessment Monitoring.

In addition to the UCMR 1 List 1 Assessment Monitoring, EPA required monitoring for selected contaminants (including fonofos) for which analytical methods were developed but not widely used. Known as the UCMR 1 List 2 Screening Survey, EPA randomly selected 300 public water systems (120 large and 180 small systems) from the pool of systems required to conduct UCMR 1 List 1 Assessment Monitoring. In total, 29,765 sample results have been collected under the UCMR 1 List 2 Screening Survey from the participating large and small systems. Approximately 2,300 samples were collected for each contaminant. The UCMR 1 List 2 Screening Survey included systems from 48 States, two U.S. Territories, and Tribal lands in one EPA Region. EPA used the occurrence data from this survey to evaluate fonofos.

EPA analyzed the UCMR 1 List 1 Assessment Monitoring and List 2 Screening Survey data to generate the following occurrence and exposure summary statistics:

- the total number of systems and the total population served by these systems,
- the number and percentage of systems with at least one observed detection that has a concentration greater than ½ the HRL and greater than the HRL (or in some cases greater than or equal to the minimum reporting level or MRL), and
- the number of people and percentage of the population served by systems with at least one observed detection greater than ½ the HRL and greater than the HRL (or in some cases greater than or equal to the MRL).[7]

The initial UCMR 1 summary occurrence statistics for dacthal mono- and di-acid degradates, DDE, 1,3-dichloropropene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, EPTC, fonofos, and terbacil are presented in Part II of this document.

Note that in some cases, for example DDE, 2,4-dinitrotoluene, and 2,6-dinitrotoluene, only an MRL analysis was performed because the MRL was higher than the HRL. EPA set the MRL for UCMR contaminants based on the capability of analytical methods, not anticipated health levels. In the case of volatile organic compounds (VOCs), the MRL was determined by multiplying by 10 either the published minimum detection limit or 0.5 µg/L, whichever was greater. For other contaminants, the MRL was determined by multiplying by 10 the least sensitive method's minimum detection limit, or, when available, multiplying by 5 the least sensitive method's estimated detection limit (USEPA, 2000c). MRLs were set approximately an order of magnitude higher than detection limits to ensure consistency, accuracy, and reproducibility of results.

[7] Both Part II of this document and EPA's technical occurrence document (USEPA, 2006b) also provide summary statistics for the median and 99th percentile concentrations of all analytical detections and detailed occurrence results based on UCMR data according to source water type (surface versus ground water), system size, and State.

The Unregulated Contaminant Monitoring (UCM) Program Rounds 1 and 2

In 1987, EPA initiated the UCM program to fulfill a 1986 SDWA Amendment that required monitoring of specified unregulated contaminants to gather information on their occurrence in drinking water for future regulatory decision-making purposes. EPA used data from the UCM program to evaluate occurrence for 2 of the 11 contaminants considered for these regulatory determination. These two contaminants are 1,3-dichloropropene and 1,1,2,2-tetrachloroethane.

EPA implemented the UCM program in two phases or rounds. The first round of UCM monitoring generally extended from 1988 to 1992 and is referred to as UCM Round 1 monitoring. The second round of UCM monitoring generally extended from 1993 to 1997 and is referred to as UCM Round 2 monitoring.

UCM Round 1 monitored for 34 VOCs, including 1,3-dichloropropene and 1,1,2,2-tetrachloroethane (52 FR 25720, July 8, 1987). UCM Round 2 monitored for 13 synthetic organic compounds (SOCs) and sulfate, and the same 34 VOCs from UCM Round 1 monitoring (57 FR 31776, July 17, 1992).

The UCM Round 1 database contains contaminant occurrence data from 38 States, Washington, DC, and the U.S. Virgin Islands. The UCM Round 2 database contains data from 34 States and several Tribes. Due to incomplete State data sets, national occurrence estimates based on raw (unedited) UCM Round 1 or Round 2 data could be skewed to low-occurrence or high-occurrence settings (e.g., some States only reported detections). To address potential biases in the data[8], EPA developed national cross-sections from the UCM Round 1 and Round 2 State data using an approach similar to that used for EPA's 1999 Chemical Monitoring Reform (CMR), the first Six Year Review, and the first CCL Regulatory Determinations. This national cross-section approach was developed to support occurrence analyses and was supported by scientific peer reviewers and stakeholders. This approach identified 24 of the original 38 States from the UCM Round 1 database and 20 of the original 34 States from the UCM Round 2 data base for the national cross-section.

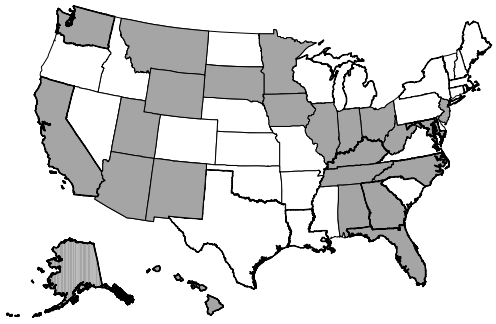
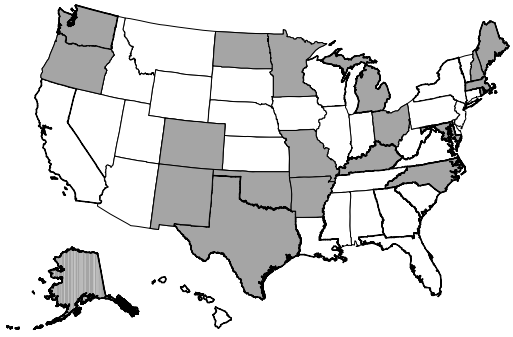
Because UCM Round 1 and Round 2 data represent different time periods and include occurrence data from different States, EPA developed separate national cross-sections for each data set. The UCM Round 1 national cross-section consists of data from 24 States, with approximately 3.3 million total analytical data points from approximately 22,000 unique PWSs. The UCM Round 2 national cross-section consists of data from 20 States, with approximately 3.7 million analytical data points from slightly more than 27,000 unique PWSs. The UCM Round 1 and 2 national cross-sections represent significantly large samples of national occurrence data. Within each cross-section, the actual number of systems and analytical records for each contaminant varies. The support document "The Analysis of Occurrence Data from the Unregulated Contaminant Monitoring (UCM) Program and National Inorganics and Radionuclides Survey (NIRS) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List" (USEPA, 2006a) provides a description of how the national

[8] The potential biases in the raw UCM data are due to lack of representativeness (since not all States provided UCM data) and incompleteness (since some States that provided data had incomplete data sets).

cross-sections for the Round 1 and Round 2 data sets were developed. Additional background information can be found in USEPA (2000b).

EPA constructed the national cross-sections in a way that provides a balance and range of States with varying pollution potential indicators, a wide range of the geologic and hydrologic conditions, and a very large sample of monitoring data points. While EPA recognizes that some limitations exist, the Agency believes that the national cross-sections do provide a reasonable estimate of the overall distribution and the central tendency of contaminant occurrence across the United States. See Exhibit 2-2 for a listing of States in each national cross-section.

Exhibit 2-3: Cross-section States for UCM Round 1 (24 States) and Round 2 (20 States)

Round 1		**Round 2**	
Alabama	Minnesota*	Alaska*	New Hampshire
Alaska*	Montana	Arkansas	New Mexico*
Arizona	New Jersey	Colorado	North Carolina*
California	New Mexico*	Kentucky*	North Dakota
Florida	North Carolina*	Maine	Ohio*
Georgia	Ohio*	Maryland*	Oklahoma
Hawaii	South Dakota	Massachusetts	Oregon
Illinois	Tennessee	Michigan	Rhode Island
Indiana	Utah	Minnesota*	Texas
Iowa	Washington*	Missouri	Washington*
Kentucky*	West Virginia		
Maryland*	Wyoming		
			

** Cross-section States in both Round 1 and Round 2*

EPA analyzed the UCM Round 1 and 2 National Cross-Section data to generate the following initial occurrence and exposure summary statistics:

- the total number of systems and the total population served by these systems,
- the number and percentage of systems with at least one observed detection that has a concentration greater than ½ the HRL and greater than the HRL (or in some cases greater than or equal to the MRL), and
- the number of people and percentage of the population served by systems with at least one observed detection that has a concentration greater than ½ the HRL and greater than the HRL (or in some cases greater than or equal to the MRL).[9]

The initial UCM summary occurrence statistics for 1,3-dichloropropene and 1,1,2,2-tetrachloroethane are presented in Part II of this document.

National Inorganics and Radionuclides Survey (NIRS)

In the mid-1980's, EPA conducted the NIRS to provide a statistically representative sample[10] of the national occurrence of inorganic contaminants in community water systems (CWSs) served by ground water. EPA used data from NIRS, as well as a supplemental survey, to evaluate occurrence for boron.

The NIRS database includes 36 radionuclides and inorganic compounds (IOCs), including boron. The NIRS provides contaminant occurrence data from 989 ground water CWSs covering 49 States (all except Hawaii) and does not include surface water systems. The survey focused on ground water systems, in part because IOCs tend to occur more frequently and at higher concentrations in ground water than in surface water. Each of the 989 randomly selected CWSs was sampled once between 1984 and 1986.

EPA analyzed the NIRS data to generate the following occurrence and exposure summary statistics for boron:

- the total number of systems and the total population served by these systems,
- the number and the percentage of systems with at least one detection that has a concentration greater than ½ the HRL and greater than the HRL,
- the number of people and percentage of the population served by systems with at least one observed detection that has a concentration greater than ½ the HRL and greater than the HRL.[11]

[9] Part II of this document and EPA's technical occurrence document (USEPA, 2006a) also provide summary statistics for the median and 99th percentile concentrations of all analytical detections and detailed occurrence results based on the UCM Round 1 and 2 National Cross-Sections according to source water type (surface versus ground water), system size, and State.

[10] NIRS was designed to provide results that are statistically representative of national occurrence at CWSs using ground water sources and is stratified based on system size (population served by the system). Most of the NIRS data are from smaller systems (92 percent from systems serving 3,300 persons or fewer).

[11] Part II of this document and EPA's UCM/NIRS technical occurrence document (USEPA, 2006a) also provide the number and percentage of systems with detections, the 99th percentile concentration of all samples, the 99th percentile concentration of samples with detections, and the median concentration of samples with detections.

Results of the NIRS analyses of boron are reported in Part II, Chapter 3. Because the NIRS data were collected in a randomly designed sample survey, these summary statistics are representative of national occurrence in ground water CWSs.

One limitation of the NIRS is a lack of occurrence data for surface water systems. To provide perspective on the occurrence of boron in surface water systems relative to ground water systems, EPA reviewed and took into consideration a recent boron occurrence survey funded by the American Water Works Association Research Foundation (AWWARF) (Frey *et al.*, 2004). A short description of the AWWARF study is provided below in Section 2.2.2, and the results of the AWWARF survey are presented in Chapter 3 (the boron chapter).

2.2.2 Supplemental Data Sources

The Agency evaluated several sources of supplemental occurrence information to augment the primary drinking water occurrence data, to evaluate the likelihood of contaminant occurrence, and/or to more fully characterize a contaminant's presence in the environment. This section provides brief descriptions of many of the supplemental information/data sources cited in Part II (and Part III) of this document.

National Center for Food and Agricultural Policy (NCFAP) Pesticide Use Database

The National Center for Food and Agricultural Policy (NCFAP), a private non-profit institution, maintains a national Pesticide Use Database. NCFAP annual pesticide use estimates for circa 1992 and circa 1997 are based on State-level commercial agriculture usage patterns for the periods 1990-1993 and 1995-1998, and State-level crop acreage for 1992 and 1997. The database contains estimates of pounds applied and acres treated in each State for 220 active ingredients and 87 crops. The majority of the chemicals monitored are herbicides, but the database also follows significant numbers of fungicides and insecticides (NCFAP, 2000).

The NCFAP database has several limitations. First, the database only includes applications of pesticides to cropland (foliar, soil, and in furrow applications). Non-cropland applications, such as uses for homes, greenhouses, livestock, or ornamentals, are not included. The database does not include non-bearing orchards or vineyards, or governmental Areawide Eradication programs. Second, in interpreting the NCFAP database, it should be noted that records are compiled from a wide variety of sources. NCFAP states that there is no way to determine the accuracy of any of the estimates in the database, adding that some are based on surveys of farmers, while others are expert opinions from knowledgeable extension service specialists. When data for particular States and crops are unavailable, as they are in many cases, values are assigned on the basis of data from a nearby State (NCFAP, 2000).

USGS Pesticide Use Maps

The United States Geological Survey has produced maps of pesticide use for 208 compounds used in U.S. crop production. The maps are based on pesticide use rates compiled by NCFAP (see NCFAP Pesticide Use Database, above). For each of the compounds, NCFAP has developed two use coefficients, the percent of acres treated for 87 specific crops and the pounds of an active ingredient applied annually to each acre of that crop. The maps combine the

NCFAP State-based pesticide use coefficients with county-level crop acreages obtained from the 1992 Census of Agriculture. This produces maps showing the distribution of average annual pesticide use. Map resolution is based at the county-level (USGS, 2004).

The maps have the same limitations in data as the NCFAP database, as described above. Additionally, the NCFAP estimates of applied pesticides are averaged at the State-level, while the maps extrapolate to the county-level by using county crop acreages from the Census of Agriculture. Consequently, the maps do not truly represent the local variability of cropping and management practices found within many States. Furthermore, the 1992 Census of Agriculture may not have represented all crop usage, nor included all types of pasture (USGS, 2004).

Toxics Release Inventory (TRI)

EPA established the Toxics Release Inventory (TRI) in 1987 in response to Section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA). EPCRA section 313 requires facilities to report to both EPA and the States annual information on toxic chemical releases from facilities that meet reporting criteria. EPCRA section 313 also requires EPA to make this information available to the public through a computer database. The database is accessible through TRI Explorer, which can be accessed at http://www.epa.gov/triexplorer. In 1990 Congress passed the Pollution Prevention Act, which required that additional data on waste management and source reduction activities be reported under TRI. The TRI database details not only the types and quantities of toxic chemicals released to the air, water, and land by facilities, but also provides information on the quantities of chemicals sent to other facilities for further management (USEPA, 2002a and 2003).

Facilities are required to report releases and other waste management activities related to TRI chemicals if they manufacture, process, or otherwise use more than established threshold quantities of these chemicals. Currently, for most chemicals the thresholds are 25,000 pounds for manufacturing and processing and 10,000 pounds for use. Both the number and type of facilities required to report has increased over time so that in 2002 over 24,000 industrial and Federal facilities submitted in excess of 93,000 reports on toxic releases. In 2000, special thresholds were added for persistent bioaccumulative toxic chemicals, for example dioxin and dioxin-like compounds (USEPA, 2002a). Today, TRI includes information on releases of nearly 670 chemicals.

Although TRI can provide a general idea of release trends, it is far from exhaustive and has significant limitations. For example, small facilities (those with fewer than 10 full-time employees and those that do not exceed manufacture and use limits) are not required to report releases. In addition, the reporting threshold for the manufacturing and processing of TRI chemicals changed between 1987 and 1989, dropping from 75,000 pounds per year in 1987 to 50,000 in 1988 to the current 25,000 in 1989; this creates the potential for misleading data trends over time (USEPA, 1996). Finally, TRI data are meant to reflect releases and should not be used to estimate general public exposure to a chemical (USEPA, 2002a).

USGS National Water Quality Assessment (NAWQA)

The USGS instituted the National Water Quality Assessment (NAWQA) program in 1991 to examine ambient water quality status and trends in the United States. The NAWQA program is designed to apply nationally consistent methods to provide a consistent basis for comparisons among study basins across the country and over time. These occurrence assessments serve to facilitate interpretation of natural and anthropogenic factors affecting national water quality. For more detailed information on the NAWQA program design and implementation, please refer to Leahy and Thompson (1994) and Hamilton *et al.* (2004).

Study Unit Monitoring

The NAWQA program conducts monitoring and water quality assessments in significant watersheds and aquifers referred to as "study units." The program's sampling approach is not "statistically" designed (i.e., it does not involve random sampling), but it provides a representative view of the nation's waters in its coverage and scope. Together, the 51 study units monitored between 1991 and 2001 include the aquifers and watersheds that supply more than 60% of the nation's drinking water and water used for agriculture and industry. The NAWQA program monitors the occurrence of chemicals such as pesticides, nutrients, VOCs, trace elements, and radionuclides, and the condition of aquatic habitats and fish, insects, and algal communities (NRC, 2002; Hamilton *et al.*, 2004). NAWQA has collected data from over 6,400 surface water and 7,000 ground water sampling points. (The NAWQA Data Warehouse can be reached via a link from the following website: http://water.usgs.gov/nawqa/data.html).

Monitoring of study units occurs in stages. Between 1991 and 2001, approximately one-third of the study units at a time were studied intensively for a period of three to five years, alternating with a period of less intensive research and monitoring that lasted between five and seven years. Thus, all participating study units rotated through intensive assessment in a ten-year cycle (Leahy and Thompson, 1994). The first ten-year cycle was designated Cycle 1. Summary reports are available for the 51 study units that underwent intensive monitoring in Cycle 1 (USGS, 2001). Cycle 2 monitoring is scheduled to proceed in 42 study units from 2002 to 2012 (Hamilton *et al.*, 2004).

USGS Analysis: National Synthesis Programs

Through a series of National Synthesis efforts, the USGS NAWQA program is preparing comprehensive analyses of data on topics of particular concern. These data are aggregated from the individual study units and other sources to provide a national overview.

Pesticide National Synthesis

The Pesticide National Synthesis began in 1991. Results from the most recent USGS Pesticide National Synthesis analysis, based on complete Cycle 1 (1991-2001) data from NAWQA study units, are posted on the NAWQA Pesticide National Synthesis website (Martin *et al.*, 2003; Kolpin and Martin, 2003; Nowell, 2003; Nowell and Capel, 2003). USGS considers these results to be provisional. Data for surface water, ground water, bed sediment, and biota are presented separately, and results in each category are subdivided by land use category. Land use

categories include agricultural, urban, mixed (deeper aquifers of regional extent in the case of ground water), and undeveloped. The National Synthesis analysis for pesticides is a first step toward the USGS goals of describing the occurrence of pesticides in relation to different land use and land management patterns, and developing a deeper understanding of the relationship between spatial occurrence of contaminants and their fate, transport, persistence, and mobility characteristics.

The surface water summary data presented by USGS in the Pesticide National Synthesis (Martin *et al.*, 2003) only include stream data. Sampling data from a single one-year period, generally the year with the most complete data, were used to represent each stream site. Sites with few data or significant gaps were excluded from the analysis. NAWQA stream sites were sampled repeatedly throughout the year to capture and characterize seasonal and hydrologic variability. In the National Synthesis analysis, the data were time-weighted to provide an estimate of the annual frequency of detection and occurrence at a given concentration.

The USGS Pesticide National Synthesis only analyzed ground water data from wells; data from springs and agricultural tile drains were not included. The sampling regimen used for wells was different than that for surface water. In the National Synthesis analysis (Kolpin and Martin, 2003), USGS uses a single sample to represent each well, generally the earliest sample with complete data for the full suite of analytes.

The NAWQA program monitored bed sediment and fish tissue at sites considered likely to be contaminated and at sites that represent various land uses within each study unit. Most sites were sampled once in each medium. In the case of sites sampled more than once, a single sample was chosen to represent the site in the Pesticide National Synthesis analysis (Nowell, 2003). In the case of multiple bed sediment samples, the earliest one with complete data for key analytes was used to represent the site. In the case of multiple tissue samples, the earliest sample from the first year of sampling that came from the most commonly sampled type of fish in the study unit was selected.

As part of the National Pesticide Synthesis, USGS also analyzed the occurrence of select semi-volatile organic compounds (SVOCs) in bed sediment at sites considered likely to be contaminated and sites that represent various land uses within each study unit (Nowell and Capel, 2003). Most sites were sampled only once. When multiple samples were taken, the earliest one was used to represent the site in the analysis.

Over the course of Cycle 1 (1991-2001), NAWQA analytical methods may have been improved or changed. Hence, reporting levels (RLs) varied over time for some compounds. In the summary tables, the highest RL for each analyte is presented for general perspective. In the ground water, bed sediment, and tissue data analyses, the method of calculating concentration percentiles sometimes varied according to how much of the data was censored at particular levels by the laboratory (i.e., because of the relatively large number of non-detections in these media).

VOC National Synthesis

The Volatile Organic Compound (VOC) National Synthesis began in 1994. The most comprehensive VOC National Synthesis reports to date are one random survey and one focused survey funded by the American Water Works Association Research Foundation (AWWARF) and carried out by USGS in collaboration with the Metropolitan Water District of Southern California and the Oregon Health & Science University. The random survey (Grady, 2003) targeted surface and ground waters used as source water by CWSs. Samples were taken from the source waters of 954 CWSs in 1999 and 2000. The random survey was designed to be nationally representative of CWS source water. In the focused survey (Delzer and Ivahnenko, 2003), 451 samples were taken from source waters serving 134 CWSs between 1999 and 2001. These surface and ground waters were chosen because they were suspected or known to contain methyl tertiary-butyl ether (MTBE). The focused survey was designed to provide insight into temporal variability and anthropogenic factors associated with VOC occurrence. Details of the monitoring plan for these two studies, including detection limits, are provided by Ivahnenko *et al.* (2001). Separately, AwwaRF also published the results of this monitoring effort (AwwaRF, 2003).

Additional products of the VOC National Synthesis include a compilation of historical VOC monitoring data from multiple studies (Squillace *et al.*, 1999). The data, collected from 2,948 wells between 1985 and 1995 by local, state, and federal agencies, were reviewed to ensure they met data quality criteria. Most of the data were from early study unit monitoring. The samples represent both urban and rural areas, and both drinking water and non-drinking water wells. A full analysis of 10 years of study unit monitoring data has not yet been performed by the VOC National Synthesis.

Trace Elements National Synthesis

A National Synthesis effort for trace elements is underway. However, the only trace element being considered for regulatory determination at this time, boron, was not included among the analytes in Cycle 1 data collection. Boron is included among the trace element analytes in NAWQA Cycle 2.

EPA Analysis of NAWQA Study Unit Monitoring Results

Whereas the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination. EPA's analysis of the NAWQA data is analogous to the simple, straight-forward "Stage 1" analysis the Agency performed on finished drinking water data from CWS monitoring. That is, all the occurrence data for a particular contaminant were compiled and analyzed using non-parametric methods to yield simple summary statistics to characterize contaminant occurrence. The analysis was performed on Cycle 1 data for DDE, DCPA, the mono-acid degradate of DCPA, EPTC, fonofos, metolachlor, MTBE, terbacil, and 1,1,2,2-tetrachloroethane. (Data were unavailable for boron, 1,3-dichloropropene, and 2,4- and 2,6-dinitrotoluene.)

The surface water data consist of stream samples; all surface water data were included in the EPA summary analysis. For ground water, all well data were used; as with the USGS National Pesticide Synthesis, data from springs and drains were excluded. For each contaminant, EPA calculated detection frequencies simply as the percentage of samples and the percentage of sites with at least one detection. (A detection is an analytical result equal to or greater than the reporting limit.) EPA used USGS data without any censoring or weighting. From samples with detects a number of descriptive statistics were also calculated, including the minimum, median, 95th percentile, 99th percentile, and maximum concentrations. Reporting limits varied over time during the NAWQA program. The highest reporting limit used for each contaminant is presented with the results of the analysis. Note that because reporting limits varied, the minimum concentration reported as a detection is often lower than the highest reporting limit. All statistics were calculated in SAS®.

USGS National Highway Runoff Data and Methodology Synthesis (USGS Stormwater Studies)

In addition to the NAWQA project, USGS has prepared additional surveys of national contaminant occurrence. For the National Highway Runoff Data and Methodology Synthesis, USGS conducted a review of 44 studies of SVOCs and VOCs in runoff conducted since 1970 (Lopes and Dionne, 1998). Most of the studies focused on SVOCs in urban stormwater and sediments. USGS evaluated the reviewed studies for data quality, including documentation of sampling protocols and methods, limits of reporting and detection, and protocols of quality-control and quality-assurance.

The Synthesis reports on a number of deficiencies in available data on highway and stormwater runoff which prevent full comparisons between studies. The greatest problem reported was that only 10 percent of the studies accurately described where in the stream cross-section study samples were taken. As SVOCs concentrate in suspended solids and suspended solids are seldom uniformly distributed in the stream profile, the absence of such data limits the reliability of findings. Another problem reported was that only 30 percent or fewer of the studies documented detection limits or quality control procedures. This limits the extent to which the findings of different studies can be compared. Finally, the report noted that many of the loading factors and regression equations used in the reviewed sources (particularly those from the 1970s) were out-of-date and needed to be readjusted if their results are to be adapted to the present day.

Of the 44 publications that the Synthesis reviews, two types of studies (encompassing several publications) deserve mention due to their wide geographic distribution. The first is the priority pollutant monitoring project of the Nationwide Urban Runoff Program (NURP). This program reported monitoring of EPA priority pollutants in 15 cities in 14 States from 1979 to 1982. The second is a set of USGS urban stormwater studies conducted in cities with a population of 100,000 or more that were required by EPA to obtain National Pollutant Discharge Elimination System (NPDES) permits. These studies involved monitoring in 16 cities in 11 States since 1991.

Pesticides in Ground Water Database (PGWDB)

The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992). Data from 68,824 wells in 45 States are included. The vast majority of the wells (65,865) were drinking water wells. Monitoring was conducted for 258 pesticides and 45 degradates. Not all studies tested for every compound.

Because PGWDB data come from multiple sources, they should be interpreted with caution. Different studies were conducted for different reasons, and used different sampling techniques and analytical methods. Detection limits were not uniform. The data are not geographically representative: results might be biased high because areas with suspected contamination are likely to have been sampled more frequently than pristine areas.

National Pesticide Survey (NPS)

In 1990, EPA completed a national survey of pesticides in drinking water wells. The purpose of the National Pesticide Survey (NPS) was to determine the national occurrence frequencies and concentrations of select pesticides in the nation's drinking water wells, and to improve EPA's understanding of how pesticide occurrence in ground water correlates with patterns of pesticide usage and ground water vulnerability. The survey included approximately 1,300 CWS wells and rural domestic wells. Sampling was conducted between 1988 and 1990. The survey targeted areas representing a variety of pesticide usage levels and ground water vulnerability. The survey was designed to provide a statistically reliable estimate of pesticide occurrence in the nation's drinking water wells. It was not designed to provide statistically valid results at the State- or local-level. Wells were sampled for 101 pesticides, 25 pesticide degradates, and nitrate (USEPA, 1990).

Community Water System Survey (CWSS)

The 2000 Community Water System Survey (CWSS) (USEPA, 2002b; 2002c) gathered data on the financial and operating characteristics of a random sample of community water systems nationwide. In addition, the Survey asked all "very large" community water systems, those that serve more than 500,000 people (a total of 83 systems), to provide monitoring results for five regulated compounds (arsenic, atrazine, 2,4-D, simazine, and glyphosate) and four unregulated compounds (radon, MTBE, metolachlor, and boron), including results from raw water at each intake and from finished water at treatment plant. EPA received completed questionnaires from 58 systems. However, not all systems answered every question. Note that because reported results are incomplete, they are more illustrative than statistically representative.

AWWARF Boron Study

The American Water Works Research Foundation funded a survey to evaluate the occurrence of boron (as well as hexavalent chromium) in drinking water sources (Frey *et al.*, 2004). The AWWARF study recruited 189 PWSs representing 407 source waters in 41 States. Of the 407 source water sample kits distributed in 2003, approximately 342 were returned. Of these 342 samples, 341 were analyzed for boron. Approximately 67 percent (or 228) represented ground water sources and 33 percent (or 113) represented surface water sources.

2.3 References

American Water Works Association Research Foundation (AwwaRF). 2003. *Occurrence of MTBE and VOCs in Drinking Water Sources of the United States.*

Delzer, G.C. and T. Ivahnenko. 2003. *Occurrence and Temporal Variability of Methyl tert-Butyl Ether (MTBE) and Other Volatile Organic Compounds in Select Sources of Drinking Water: Results of the Focused Survey.* U.S. Geological Survey Water-Resources Investigations Report 02-4084, 65 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4084.pdf.

Frey, M.M., C. Seidel, M. Edwards, J. Parks, and L. McNeill. 2004. *Occurrence Survey for Boron and Hexavalent Chromium.* AwwaRF Report 91044F.

Grady, S.J. 2003. *A National Survey of Methyl tert-Butyl Ether and Other Volatile Organic Compounds in Drinking-Water Sources: Results of the Random Survey.* U.S. Geological Survey Water-Resources Investigations Report 02-4079, 85 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4079.pdf.

Hamilton, P.A., T.L. Miller, and D.N. Myers. 2004. *Water Quality in the Nation's Streams and Aquifers: Overview of Selected Findings, 1991-2001*. USGS Circular 1265. Available on the Internet at: http://water.usgs.gov/pubs/circ/2004/1265/pdf/circular1265.pdf.

Ivahnenko, T., S.J. Grady, and G.C. Delzer. 2001. *Design of a National Survey of Methyl tert-Butyl Ether and Other Volatile Organic Compounds in Drinking-Water Sources.* U.S. Geological Survey Open-File Report 01-271, 42 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr01_271.pdf.

Kolpin, D.W. and J.D. Martin. 2003. "Pesticides in Ground Water: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestgw/Pest-GW_2001_Text.html. Accessed August 24, 2004.

Leahy, P.P. and T.H. Thompson. 1994. *The National Water-Quality Assessment Program*. U.S. Geological Survey Open-File Report 94-70, 4 p. Available on the Internet at: http://water.usgs.gov/nawqa/NAWQA.OFR94-70.html. Accessed October 25, 2004.

Longtin, J.P. 1988. Occurrence of radon, radium and uranium in groundwater. *Journal of the American Water Works Association*. 80(7):84-93 (July).

Lopes, T.J. and S.G. Dionne. 1998. *A Review of Semivolatile and Volatile Organic Compounds in Highway Runoff and Urban Stormwater.* U.S. Geological Survey Open-File Report 98-409, 67 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr98-409.pdf.

Martin, J.D., C.G. Crawford, and S.J. Larson. 2003. "Pesticides in Streams: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestsw/Pest-SW_2001_Text.html. Accessed August 24, 2004.

National Center for Food and Agricultural Policy (NCFAP). 2000. *Pesticide Use in U.S. Crop Production: 1997. National Summary Report.* Available on the Internet at: http://www.ncfap.org/ncfap/nationalsummary1997.pdf.

National Research Council (NRC). 2002. *Opportunities to Improve the U.S. Geological Survey National Water Quality Assessment Program.* National Academy Press. 238 p. Available on the Internet at: http://www.nap.edu/catalog/10267.html.

Nowell, L. 2003. "Organochlorine Pesticides and PCBs in Bed Sediment and Aquatic Biota from United States Rivers and Streams: Summary Statistics; Preliminary Results of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/sedbiota/. Accessed August 25, 2004. Last updated April 10, 2003.

Nowell, L. and P. Capel. 2003. "Semivolatile Organic Compounds (SVOC) in Bed Sediment from United States Rivers and Streams: Summary Statistics; Preliminary Results of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/svoc/SVOC-SED_2001_Text.html. Accessed August 25, 2004. Last updated May 12, 2003.

Squillace, P.J., M.J. Moran, W.W. Lapham, C.V. Price, R.M. Clawges and J.S. Zogorski. 1999. Volatile organic compounds in untreated ambient groundwater of the United States, 1985-1995. *Environmental Science and Technology*. 33(23):4176-4187. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/journal/EST.voc.squillace.pdf.

USEPA. 1986. EPA Guidelines for Carcinogen Risk Assessment. *Federal Register*. Vol. 51, No. 185. p. 33992, September 24, 1986.

USEPA. 1990. *National Pesticide Survey: Survey Analytes.* EPA Report 570-9-90-NPS2. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS2.]

USEPA. 1992. *Pesticides in Ground Water Database: A Compilation of Monitoring Studies, 1971-1991. National Summary.* Office of Prevention, Pesticides and Toxic Substances. EPA Report 734-12-92-001. Available on the Internet at: http://nepis.epa.gov/pubtitleOPPTS.htm. [Search for document number 7341292001.]

USEPA. 1996. *1996 Toxics Release Inventory: Public Data Release Report. Chapter 3: Year-to-Year Comparison of Toxics Release Inventory Data*. Available on the Internet at: http://www.epa.gov/tri/tridata/tri96/pdr/chap3.pdf.

USEPA. 1998. *Reregistration Eligibility Decision (RED)--1,3-Dichloropropene*. EPA Report 738-R-98-016. Washington, DC: Office of Prevention, Pesticides and Toxic Substances. December 1998. Available on the Internet at: http://www.epa.gov/oppsrrd1/REDs/0328red.pdf.

USEPA. 1999. *Guidelines for Carcinogen Risk Assessment.* NCEA-F-0644, Review Draft. July 1999. Available on the Internet at: http://www.epa.gov/iris/cancer_gls.pdf.

USEPA. 2000a. "Integrated Risk Information Systems (IRIS), 1,3-Dichloropropene." May 2000. Available on the Internet at: http://www.epa.gov/iris/subst/0224.htm. Accessed February 2, 2005.

USEPA. 2000b. *Occurrence of Unregulated Contaminants in Public Water Systems: An Initial Assessment*. EPA Report 815-P-00-001.

USEPA. 2000c. *Unregulated Contaminant Monitoring Regulation Analytical Methods and Quality Control Manual*. EPA Report 815-R-00-006.

USEPA. 2001a. Notice of opportunity to provide additional information and comment on draft revised Guidelines for Carcinogen Risk Assessment (July 1999), availability of draft revised Guidelines, and adoption of draft revised Guidelines as interim guidance. *Federal Register.* Vol. 66, No. 230. p. 59593, November 29, 2001.

USEPA. 2001b. *Reference Guide for the Unregulated Contaminant Monitoring Regulation*. EPA 815-R-01-023. Office of Water, USEPA. 65 pp. Available on the Internet at: http://www.epa.gov/safewater/standard/ucmr/pdfs/ref_guide.pdf.

USEPA. 2002a. *The Toxics Release Inventory (TRI) and Factors to Consider When Using TRI Data*. EPA Report 260-F-02-017. November 2002. Available on the Internet at: http://www.epa.gov/triinter/2002_tri_brochure.pdf.

USEPA. 2002b. *Community Water System Survey 2000. Volume I: Overview*. EPA Report 815-R-02-005A. December 2002. Available on the Internet at: http://www.epa.gov/safewater/consumer/pdf/cwss_2000_volume_i.pdf.

USEPA. 2002c. *Community Water System Survey 2000. Volume II: Detailed Tables and Survey Methodology*. EPA Report 815-R-02-005B. Available on the Internet at: http://www.epa.gov/safewater/consumer/pdf/cwss_2000_volume_ii.pdf.

USEPA. 2003. *How are the Toxics Release Inventory Data Used?* EPA Report 260-R-002-004. May 2003. Available on the Internet at: http://www.epa.gov/tri/guide_docs/2003_datausepaper.pdf.

USEPA. 2005a. *Guidelines for Carcinogen Risk Assessment.* EPA Report 630-P-03-001B. March 2005. Available on the Internet at: http://www.epa.gov/ttn/atw/cancer_guidelines_final_3-25-05.pdf.

USEPA. 2005b. *Supplemental Guidance for Assessing Susceptibility to Early-life Exposure to Carcinogens.* EPA Report 630-R-03-003F. March 2005. Available on the Internet at: http://www.epa.gov/ttn/atw/childrens_supplement_final.pdf.

USEPA. 2005c. *Application of New Cancer Guidelines.* Memorandum from Acting Administrator Stephen L. Johnson. March 29, 2005.

USEPA. 2006a. *The Analysis of Occurrence Data from the Unregulated Contaminant Monitoring (UCM) Program and National Inorganics and Radionuclides Survey (NIRS) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-009. Draft. December 2006.

USEPA. 2006b. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

United States Geological Survey (USGS). 2001. "Summary publications from 51 NAWQA study units sampled in 1991-2001." Available on the Internet at: http://water.usgs.gov/nawqa/nawqasum/. Accessed May 24, 2004. Last updated May 19, 2004.

USGS. 2004. Sources and Limitations of Data Used to Produce Maps of Annual Pesticide Use. Available on the Internet at: http://ca.water.usgs.gov/pnsp/use92/mapex.html. Accessed October 25, 2004.

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

Part II: CCL 2 Contaminants Undergoing Regulatory Determination

Chapter 3:
Boron

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

Boron, an inorganic compound (IOC), is a non-volatile metalloid that is ubiquitous in the environment in compounds called borates. Common borates include boron oxide, boric acid, and borax. Anthropogenic boron compounds include boron halides (e.g., boron trichloride and boron trifluoride). Borates and other boron compounds are used in the production of glass, ceramics, soaps, fire retardants, pesticides, cosmetics, photographic materials, and high energy fuels.

Boron enters the environment mainly through the weathering of rocks, boric acid volatilization from seawater, and volcanic and geothermal activity. To a lesser extent, boron is released to the environment from anthropogenic sources (e.g., via industrial air emissions, fertilizer and herbicide applications, and industrial and municipal wastes). Limited data are available on the quantity of anthropogenic releases. Toxic Release Inventory (TRI) data suggest that air emissions dominate industrial boron trihalide releases. Boron trichloride releases fluctuate in the range of hundreds of pounds per year, and boron trifluoride releases fluctuate in the range of tens of thousands of pounds per year. Around 1990, the quantity of boron minerals used annually for agricultural purposes was estimated to have been approximately 293,000 pounds.

The Institute of Medicine of the National Academies categorizes boron as a possible trace mineral nutrient for humans. It may interact with Vitamin D and calcium homeostasis, influence estrogen metabolism, and play a role in cognitive function. The estimated average dietary intake of boron in U.S. male adults is 1.5 mg/day. Large doses (on the order of 20 mg/kg or more) can cause nausea and vomiting. Chronic low-level oral exposure causes developmental defects in animal subjects. Based on developmental defects in rats, the EPA reference dose (RfD) for boron is 0.2 mg/kg/day. EPA calculated a health reference level (HRL) of 1.4 mg/L or 1,400 µg/L for boron using the RfD of 0.2 mg/kg-day and a 20 percent screening relative source contribution (RSC). Sensitive subpopulations may include developing fetuses and individuals with impaired kidney function.

EPA evaluated boron occurrence in drinking water using data collected from 989 ground water public water systems (PWSs) by the National Inorganics and Radionuclides Survey (NIRS). The NIRS data indicate that approximately 4.3 percent of the ground water PWSs had detections of boron at levels greater than 700 µg/L (1/2 the HRL), affecting approximately 2.9 percent of the population served by these ground water systems. Approximately 1.7 percent of the ground water PWSs had detections of boron at levels greater than 1,400 µg/L (the HRL), affecting approximately 0.4 percent of the population served by these ground water systems.

Because NIRS only investigated ground water systems, the Agency evaluated the results of a survey funded by the American Water Works Association Research Foundation (AWWARF) to gain a better understanding of the potential occurrence of boron in surface water systems. Of 341 samples analyzed for boron, approximately 67 percent represented ground water sources and 33 percent represented surface water sources. Of the ground water sources, 3.1% had boron concentrations that exceeded the HRL of 1,400 µg/L; the highest observed concentration was approximately 3,300 µg/L. In contrast, none of the surface water sources

exceeded the boron HRL of 1,400 µg/L, and the highest concentration in surface water was 345 µg/L. These results indicate that boron contamination occurs less frequently and at lower concentrations in surface water than in ground water.

EPA evaluated supplementary data on boron occurrence in ambient and drinking water from additional sources, including the United States Geological Survey (USGS) National Ambient Water Quality Assessment (NAWQA) program and the Community Water System Survey (CWSS).

The Agency has made a preliminary determination not to regulate boron with a national primary drinking water regulation (NPDWR). While boron was found at levels greater than the HRL (and ½ the HRL) in several of the ground water systems surveyed by NIRS, it was not found at levels greater than the HRL (or ½ the HRL) in the surface waters sources evaluated in the AWWARF study. Taking this surface water information into account, the Agency believes that the overall national occurrence and exposure from both surface and ground water systems together is likely to be lower than the values observed for the NIRS ground water data. Because boron is not likely to occur at levels of concern when considering both surface and ground waters systems, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

The Agency encourages those States with public water systems that have boron at concentrations above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory for boron to provide more recent health information. The updated Health Advisory will provide information to any States with public water systems that may have boron above the HRL.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

AWWARF	American Water Works Association Research Foundation
BMD	Benchmark Dose
CAS	Chemical Abstracts Service
CCL 2	Second Contaminant Candidate List
CWSS	Community Water Systems Survey
HF	Hollow-Fiber
HRL	Health Reference Level
ICP	Inductively Coupled Plasma
ICP-AES	Inductively Coupled Plasma-Atomic Emission Spectrometry
IOC	Inorganic Compound
IOM	Institute of Medicine
LOAEL	Lowest Observed Adverse Effect Level
MDL	Method Detection Limit
MRL	Minimum Reporting Level
MTBE	Methyl tertiary-butyl ether
NAWQA	National Water Quality Assessment
NIRS	National Inorganics and Radionuclides Survey
NOAEL	No Observed Adverse Effect Level
NPDWR	National Primary Drinking Water Regulation
PTFE	Polytetrafluoroethylene
PWS	Public Water System
RfD	Reference Dose
RO	Reverse Osmosis
RSC	Relative Source Contribution
SM	Standard Method
SW	Spiral-Wound
TRI	Toxics Release Inventory
USGS	United States Geological Survey

3 Boron

3.1 Definition

Boron, an inorganic compound (IOC), is a non-volatile metalloid that is ubiquitous in the environment in compounds called borates. Common borates include boron oxide, boric acid, and borax. Anthropogenic boron compounds include boron trichloride and boron trifluoride. The Chemical Abstracts Service (CAS) registry number of elemental boron is 7440-42-8. Borates and other boron compounds have their own registry numbers.

3.1.1 Properties and Sources

Elemental boron is not readily found in nature, yet borates are natural and ubiquitous. Elemental boron exists as a solid at room temperature, either as black monoclinic crystals or as a yellow or brown amorphous powder when impure. Boron is an essential nutrient for plants and an essential element for many organisms (USEPA, 1994a). Borates are most predominantly found in nature in oceans, sedimentary rocks, coal, shale, and some soils. Boron enters the environment mainly through the weathering of rocks, boric acid volatilization from seawater, and volcanic and geothermal activity (HSDB, 2004; ATSDR, 1992). To a lesser extent, boron is also released to the environment through anthropogenic sources. Anthropogenic boron compounds include boron halides (boron trichloride and boron trifluoride) as well as borates. Boron compounds are used in the production of glass, ceramics, soaps, fire retardants, pesticides, cosmetics, photographic materials, and high energy fuels (USGS, 2004; ATSDR, 1992). Boron compounds are registered as pesticides in the U.S. for use as insecticides, herbicides, and fungicides. In such roles, boron compounds can act in a number of ways, such as through poisoning, desiccation, or inhibition of growth (USEPA, 1994a). The production and use of products containing boron compounds adds to the release of boron into the environment. Physical and chemical properties of boron and selected boron compounds are summarized in Exhibit 3-1.

Exhibit 3-1: Physical and Chemical Properties

Identification: Boron and Boron Compounds							
	boron	***boron oxide***	***boric acid***	***borax***	***borax, anhydrous***	***boron trichloride***	***boron trifluoride***
CAS number	7440-42-8	1303-86-2	10043-35-3	1303-96-4	1303-96-4	10294-34-5	7637-07-02
Molecular Formula	B	B_2O_3	H_3BO_3	$Na_2B_4O_7 \cdot 10H_2O$	$Na_2B_4O_7$	BCl_3	BF_3
Physical and Chemical Properties							
Boiling Point	2,550 °C [1]	1,500 °C [1]	$-1^{1/2}$ H_2O, 300 °C [1]	$-10H_2O$, 320 °C [2]	Decomposes at 1,575 °C [1]	12.5 °C [2]	-99.9 °C [1]
Melting Point	2,300 °C [1]	450 ± 2 °C [1]	169 ± 1 °C tr to HBO_2 [1]	75 °C, $-8H_2O$, 60 °C [2]	741 °C [1]	-107 °C [2]	-126.8 °C [1]
Molecular Weight	10.81 /mol[2]	69.64 g/mol [2]	61.84 g/mol [2]	381.37 g/mol [1]	201.22 g/mol [1]	117.19 g/mol [2]	67.81 g/mol [2]
Log K_{oc}	-----	-----	-----	-----	-----	-----	-----
Log K_{ow}	-----	-----	-----	-----	-----	-----	-----
Water Solubility	insoluble [2]	rapidly hydrates to boric acid [1]	63.5 g/L at 30 °C [1]	20.1 g/L at 0 °C [1]	10.6 g/L at 0 °C; 87.9 g/L at 40 °C [1]	decomposes [1]	1060 g/L at 20 °C [2]
Vapor Pressure	1.56×10^{-5} atm at 2,140 °C [2]	-----	-----	-----	-----	100 mm Hg at 33.5 °C [1]	40 mm Hg at -131°C (solid); 760 mm Hg at -110.7 °C (liq) [1]
Henry's Law Constant	-----	-----	-----	-----	-----	-----	-----
Freundlich Isotherm Constant (K)	-----	-----	-----	-----	-----	-----	-----

[1] *Weast, 1988 (as cited in ATSDR, 1992 and HSDB, 2004)*
[2] *Budavari, 1989 (as cited in HSDB, 2004)*

3.1.2 Environmental Fate and Behavior

Little is known about the residence time of boron compounds in air, soil, or water. Limited data are available regarding their transport and partitioning in these media (ATSDR, 1992).

The adsorption of borates and boric acids to soils is controlled by the presence of aluminum and iron oxides and, to a lesser extent, organic matter (Bingham *et al.*, 1971; Sakata, 1987; Parks and White, 1952 all as cited in ATSDR, 1992). Soils rich in these oxides, like the Ultisols of the southeastern United States, will experience significant adsorption of available borates. In some environments, adsorption to soil particles may be irreversible (Rai *et al.*, 1986 as cited in ATSDR, 1992). Boron is found in soil (as borates) at an average concentration of 10 mg/kg (Weast, 1988 as cited in HSDB, 2004).

When released to the atmosphere, borates exist as particulate matter or aerosols, with a half-life on the order of days, depending on particle size and atmospheric conditions. Deposition can occur through dryfall, and wet deposition is also possible in the case of more soluble borates (USEPA, 1987 as cited in ATSDR, 1992).

In water, boron readily hydrolyzes and may polymerize in concentrated solutions (ATSDR, 1992). Adsorption to sediments is thought likely to be the most significant fate pathway for boron in water (Rai *et al.*, 1986 as cited in ATSDR, 1992). The extent of adsorption is determined by the pH of the water and the chemical composition of the sediment. The greatest adsorption takes place in a pH range of 7.5 to 9.0 (Keren *et al.*, 1981; Keren and Mezuman, 1981; Waggott, 1969 all as cited in ATSDR, 1992). Boron compounds in water may also co-precipitate as hydroxyborate compounds with aluminum, iron, or silicon (Biggar and Fireman, 1960 as cited in ATSDR, 1992). Boron is typically found in salt water at concentrations of 4.6 mg/L (Weast, 1988 in HSDB, 2004).

Aquatic plants and animals accumulate boron, but residues do not increase through the food chain (Moore, 1991). Even though it is found in many fruits and vegetables, boron does not accumulate in human tissues (Butterwick *et al.*, 1989 as cited in ATSDR, 1992; Waggot, 1969 as cited in ATSDR, 1992).

3.2 Health Effects

The Institute of Medicine (IOM, 2001) of the National Academies categorizes boron as a possible trace mineral nutrient for humans. Boron is essential for plant growth and deficiency studies in animals and humans have provided some evidence that low intakes of boron affects cellular function and the activity of other nutrients. It may interact with Vitamin D and calcium homeostasis, influence estrogen metabolism, and play a role in cognitive function (IOM, 2001). Iyengar *et al.* (1988, as cited in USEPA, 2004a) reported an average dietary intake of 1.5 mg/day for male adults based on the Food and Drug Administration Total Diet Study.

Some human oral data are available from cases where boron was ingested as a medical treatment. When the amount ingested was less than 3.68 mg/kg, subjects were asymptomatic, while doses of 20 and 25 mg/kg resulted in nausea and vomiting. Case reports and surveys of

accidental poisonings indicate that the lethal doses of boron range from 15 to 20 grams (approximately 200 to 300 mg/kg) for adults, 5 to 6 grams (approximately 70 to 85 mg/kg) for children, and 2 to 3 grams (approximately 30 to 45 mg/kg) for infants (USEPA, 2004b).

The primary adverse effects seen in animals after chronic exposure to low doses of boron generally involve the testes and developing fetus. Chronic effects of dietary boron exposure in two-year studies included testicular atrophy and spermatogenic arrest in dogs, decreased food consumption, suppressed growth, and testicular atrophy in rats, and decreased survival, testicular atrophy, and interstitial cell hyperplasia in mice. Although researchers observed some increases in tumor incidences in the liver and in subcutaneous tissues in mice, based on comparisons to historic controls, these tumors were determined not to be associated with exposure to boron from boric acid (USEPA, 2004b). Boron is not considered mutagenic and the Agency determined that there are inadequate data to assess the human carcinogenic potential for boron (USEPA, 2004a).

In developmental studies with rats, mice, and rabbits, oral exposure to boric acid resulted in decreased pregnancy rate, increased prenatal mortality, decreased fetal weights, and increased malformations in fetuses and pups. However, these reproductive effects were associated with maternal toxicity including changes in maternal organ weights, body weights, weight gain, and increased renal tubular dilation and/or regeneration (Price *et al.*, 1990, 1994, 1996; Heindel *et al.*, 1992, 1994; Field *et al.*, 1989; all as cited in USEPA, 2004b). Reproductive effects in males were noted in the subchronic and chronic studies described in the preceding paragraphs.

The EPA reference dose (RfD) for boron is 0.2 mg/kg/day (USEPA, 2004a) based on developmental effects in rats from two studies (Price *et al.*, 1996; Heindel *et al.*, 1992; both as cited in USEPA, 2004b). The RfD was derived using the benchmark dose (BMD) method ($BMDL_{05}$ from Allen *et al.*, 1996 as cited in USEPA, 2004b). EPA calculated the health reference level (HRL) of 1.4 mg/L or 1,400 µg/L for boron using the RfD of 0.2 mg/kg/day and a 20 percent screening relative source contribution.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. Studies in rats, mice, and rabbits identify the developing fetus as potentially sensitive to boron. Price *et al.* (1996 as cited in USEPA, 2004b) identified a "lowest-observed-adverse-effect level" (LOAEL) of 13.3 mg/kg-day and a "no-observed-adverse-effect level" (NOAEL) of 9.6 mg/kg-day in the developing fetus, based on decreased fetal body weight in rats. Accordingly, boron at concentrations greater than the HRL might have an effect on prenatal development. Individuals with severely impaired kidney function might also be sensitive to boron exposure since the kidney is the most important route for excretion.

3.3 Occurrence and Exposure

3.3.1 Use and Environmental Release

The major commercial uses of boron are in the production of glass and ceramics. According to the United States Geological Survey (USGS), in 2003 these industries accounted for 78 percent of U.S. consumption of boron compounds. The industries were primarily located in the North Central United States and Eastern United States. Soaps and detergents accounted for an additional 6 percent of U.S. consumption, agriculture accounted for 4 percent, fire retardants accounted for 3 percent, and other uses accounted for 9 percent (USGS, 2004). Experimental uses include recyclable sodium borohydride fuel for powering fuel cell vehicles (USGS, 2004). Borax and boric acid are used as a neutron absorber in atomic reactors; other miscellaneous uses of borates are found in cosmetics and leather tanning (ATSDR, 1992). Boron trichloride is used in the manufacture and purification of boron, for catalysis of organic reactions, in semiconductors, in purification of metal alloys, and in bonding of iron and steels. Boron trifluoride is used predominately in catalysis, but is also used as a fumigant, in metallurgy, and for neutron detection (Windholz *et al.*, 1983).

According to the website of Rio Tinto Borax, the largest commercial producer of borates, world demand for borates is distributed among the following major uses: insulation fiberglass, textile fiberglass, and heat-resistant glass account for 42 percent of world demand; ceramic tile bodies and ceramic and enamel frits and glazes account for 14 percent; detergents, soaps, and personal care products account for approximately 10 percent; agricultural micronutrients account for 7 percent; and other uses, including wood preservatives, flame retardants, and pest control products, account for 27 percent (Rio Tinto Borax, 2004).

The United States, Turkey, and Russia are the leading producers of boron compounds, followed by Argentina, Chile, and China (USGS, 2004). In 2003, Turkey produced the greatest quantity of ore, while the U.S. led in production of refined boron compounds. U.S. boron resources, mostly sediments and brines, are primarily located in California. U.S. production of boron compounds between 1999 and 2003 ranged between 518,000 metric tons and 618,000 metric tons (measured as boric oxide). In 2003, the U.S. imported approximately 174,000 metric tons of boron compounds and exported approximately 244,000 metric tons (USGS, 2004).

Environmental boron can have natural or anthropogenic sources. Boron is a naturally occurring compound, usually found in inorganic form in sediments and sedimentary rocks. Natural weathering of rocks is thought to be the primary source of boron compounds in water and soil (Butterwick *et al.*, 1989 as cited in ATSDR, 1992). Such weathering varies geographically, however. In the United States, the richest deposits are in California. Releases to air from oceans, volcanos, and geothermal steam are other natural sources of boron in the environment (Graedel, 1978 as cited in ATSDR, 1992). Global releases of elemental boron through weathering, volcanic, and geothermal processes are estimated at approximately 360,000 metric tons annually (Moore, 1991). Human causes of boron contamination include releases to air from power plants, chemical plants, and manufacturing facilities. Fertilizers, herbicides, and industrial wastes are among the sources of soil contamination. Contamination of water can come directly from industrial wastewater and municipal sewage, as well as indirectly from air

deposition and soil runoff (ATSDR, 1992). Borates in detergents, soaps, and personal care products can also contribute to the presence of boron in water.

Boric acid and its sodium salts are registered for use as pesticides. Data from the U.S. Bureau of Mines, cited in EPA's 1994 reregistration eligibility document for boron pesticides (USEPA, 1994a), suggest that approximately 293,000 pounds of boron minerals were used annually for "agricultural purposes" around 1990. In the reregistration eligibility document EPA stated that the amount of boron used specifically as pesticide is somewhat less than the amount used for agricultural purposes, and that boric acid use declined significantly during the 1980s (USEPA, 1994a).

Two anthropogenic boron compounds, boron trichloride and boron trifluoride, are listed as Toxics Release Inventory (TRI) chemicals. For a discussion of the nature and limitations of TRI data, see Chapter 2.

TRI data for boron trichloride (see Exhibit 3-2) are reported for the years 1995 to 2003 (USEPA, 2006a). For boron trichloride, on-site air emissions constitute all of the total releases (on- and off-site), and these have generally fluctuated in the range of hundreds of pounds per year during the period of record. TRI releases for boron trichloride were reported from facilities in 6 States (Arizona, California, Indiana, New Mexico, Pennsylvania, and Wisconsin).

Exhibit 3-2: Environmental releases (in pounds) of boron trichloride in the United States, 1995-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges[1]	Underground Injection	Releases to Land		
1995	5	-	0	0	0	5
1996	37	-	0	0	0	37
1997	754	0	0	0	0	754
1998	750	0	0	0	0	750
1999	350	-	0	0	0	350
2000	605	-	0	0	0	605
2001	626	0	0	0	0	626
2002	258	0	0	0	0	258
2003	5	-	0	0	0	5

[1] *"-" denotes blank cells on reporting forms. "0" is entered when the reporting forms contained only zeros or "NA"s.*

Source: USEPA, 2006a.

Boron trifluoride releases, also for the years 1995 to 2003 (see Exhibit 3-3), are similarly dominated by on-site air emissions. Boron trifluoride releases ranged in the tens of thousands of pounds annually. TRI releases for boron trifluoride were reported from facilities in 14 States (AL, AR, DE, FL, KY, LA, MD, NY, OH, OK, PA, SC, TN, and TX) (USEPA, 2006a).

Exhibit 3-3: Environmental releases (in pounds) of boron trifluoride in the United States, 1995-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1995	25,019	0	0	0	929	25,948
1996	29,881	0	0	0	0	29,881
1997	21,290	0	0	0	5	21,295
1998	37,802	5	0	0	0	37,807
1999	16,725	0	0	0	0	16,725
2000	11,595	0	0	0	250	11,845
2001	11,496	0	0	0	0	11,496
2002	10,114	0	0	0	0	10,114
2003	7,513	0	0	0	4,295	11,808

Source: USEPA, 2006a.

3.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are the source of most drinking water. Data on the occurrence of boron in ambient water, as well as biotic tissue and bed sediment, are available from the National Water Quality Assessment (NAWQA) program of the USGS. For more information on this program, see Chapter 2. The Minnesota Pollution Control Agency has also surveyed boron in ground water.

NAWQA

No national NAWQA data are available on the occurrence of boron in ambient waters. However, some regional data are available. Boron was among the analytes in USGS ground water monitoring in the Sacramento Valley in California in 1996 (Dawson, 2001) and the lower Illinois River Basin from 1984 to 1991 (Warner, 1999). Boron was also an analyte in NAWQA studies of bed sediments and/or fish tissues from the Tualatin River Basin of Oregon from 1992 and 1996 (Bonn, 1999), the Lower Snake River Basin of Idaho and Oregon in 1997 (Clark and Maret, 1998), and the Yellowstone River Basin in Montana, North Dakota, and Wyoming from 1976 to 1979 (Peterson and Zelt, 1999).

In ground water from the Sacramento Valley aquifer, boron was detected in all 31 samples, in concentrations ranging from 12 µg/L to 1,100 µg/L. The median concentration was 42 µg/L. Two of the 31 samples had concentrations in excess of the then-current Health Advisory Level of 600 µg/L (Dawson, 2001). (That lifetime Health Advisory Level was associated with an RfD of 0.09 mg/kg/day. In June 2004 EPA established the current RfD of 0.02 mg/kg/day.)

In the lower Illinois River Basin, 71 percent of ground water samples collected between 1984 and 1991 contained boron concentrations higher than the minimum reporting level for this study of 50 µg/L. The highest detected concentration was 2,100 µg/L. Higher boron concentrations were generally found in deeper and more ancient aquifers (Warner, 1999).

At a minimum reporting level of 0.2 µg/g dry weight, boron was detected in 100 percent of ten fish tissue samples from the Tualatin River Basin. The median concentration was 1.2 µg/g and the maximum concentration was 3.5 µg/g (Bonn, 1999).

At a minimum reporting level of 0.1 µg/g dry weight, boron was detected in most or all of 25 fish tissue samples from the Lower Snake River Basin, in concentrations as high as 1.8 µg/g (Clark and Maret, 1998).

At a reporting limit of 10 mg/kg, boron was detected in 98 percent of bed sediment samples in the Yellowstone River Basin. The median concentration was 28 mg/kg and the 95th percentile concentration was 57 mg/kg (Peterson and Zelt, 1999).

Minnesota Pollution Control Agency

As a baseline survey of ground water quality in the State, the Minnesota Pollution Control Agency took samples from 954 drinking water wells between 1992 and 1996. Seventy (8.7 percent of) samples had boron concentrations in excess of 600 µg/L, and another 25 samples had concentrations between 500 and 600 µg/L. High boron concentrations in Cretaceous, Precambrian, and buried Quaternary aquifers are likely due to the natural abundance of boron in the earth's crust. High boron concentrations in surficial Quaternary aquifers, on the other hand, are likely due to anthropogenic factors. The overall median concentration of boron was 46 µg/L (MPCA, 1998).

3.3.3 Drinking Water Occurrence

In the 1980s, EPA collected nationally representative data on boron occurrence in drinking water from public water systems served by ground water as part of the National Inorganics and Radionuclides Survey (NIRS). More recently, the American Water Works Association Research Foundation (AWWARF) conducted a study of boron occurrence that included both ground water and surface water systems.

NIRS

Between 1984 and 1986, single samples were taken from 989 community public water systems (PWSs) under NIRS. Only systems served by ground water were included in the survey. Systems were selected to be geographically representative, and to include a representative distribution of system sizes. For more details on NIRS, see Chapter 2 and USEPA (2006b).

Results of the survey are presented in Exhibit 3-4. Approximately 81.9 percent of groundwater PWSs had detections of boron (that is, concentrations at or above the minimum reporting level: ≥ MRL, or ≥ 0.005 mg/L). These detections affected about 88.1 % of the population served by the PWSs, equivalent to approximately 75.5 million people served by ground water nationally. Detections at a concentration greater then one-half the HRL (> ½ HRL, or > 0.7 mg/L) occurred in 4.3% of surveyed PWSs, affecting 2.9% of the population served, equivalent to approximately 2.5 million people nationally. Concentrations greater than the HRL (> HRL, or > 1.4 mg/L) were found in approximately 1.7% of surveyed PWSs,

affecting 0.4% of the population served, equivalent to approximately 0.4 million people nationally.

Exhibit 3-4: Summary NIRS Occurrence Statistics for Boron in Ground Water Systems

Frequency Factors	NIRS Data on Boron		National System & Population Numbers[1]
Total Number of Ground Water Samples/Systems	989		59,440
99th Percentile Concentration (all samples)	2.44 mg/L		--
Health Reference Level (HRL)	1.4 mg/L		--
Minimum Reporting Level (MRL)	0.005 mg/L		--
Maximum Concentration of Detections	3.95 mg/L		--
99th Percentile Concentration of Detections	2.6 mg/L		--
Median Concentration of Detections	0.047 mg/L		--
Total Population Served by Ground Water	1,482,153		85,681,696
Occurrence by Sample/System	**Number**	**Percentage**	**National Extrapolation**
Ground Water PWSs with Detections (≥ MRL)	810	81.9%	48,682
Range of NIRS States	0 - 74	0 - 100%	N/A
Ground Water PWSs > 1/2 HRL	43	4.3%	2,584
Range of NIRS States	0 - 8	0 - 37%	N/A
Ground Water PWSs > HRL	17	1.7%	1,022
Range of NIRS States	0 - 5	0 - 26%	N/A
Occurrence by Population Served			
Population Served by GW PWSs with Detections	1,306,048	88.1%	75,501,000
Range of NIRS States	0 - 343,465	0 - 100%	N/A
Population Served by GW PWSs > 1/2 HRL	42,702	2.9%	2,469,000
Range of NIRS States	0 - 20,465	0 - 34%	N/A
Population Served by GW PWSs > HRL	6,443	0.4%	372,000
Range of NIRS States	0 - 2,500	0 - 34%	N/A

1. Total PWS and population numbers are from EPA's March 2000 Water Industry Baseline Handbook, 2nd Edition. National extrapolations are generated by multiplying the system/population percentages and the national Baseline Handbook system/population numbers.

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; N/A = Not Applicable; Total Number of Samples/Systems = total number of samples/systems on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Population Served = the total population served by PWSs for which sampling results are available; Ground Water PWSs with Detections, PWSs > ½ HRL, or PWSs > HRL = GW PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by GW PWSs with Detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by GW PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

AWWARF Boron Study

Both ground water PWSs and surface water PWSs were included in a boron survey funded by the American Water Works Research Foundation (Frey *et al.*, 2004). The AWWARF study recruited 189 PWSs representing 407 source waters in 41 States. Of the 407 source water sample kits distributed in 2003, approximately 342 were returned. Of these 342 samples, 341 were analyzed for boron. Approximately 67 percent (or 228) represented ground water sources and 33 percent (or 113) represented surface water sources. The samples were analyzed for boron with a method detection limit of 2.0 µg/L (Frey *et al.*, 2004).

Boron was detected with concentrations equal or greater than the method detection limit in 226 of 228 ground water samples (99.1%) and 110 of 113 surface water samples (97.3%). Boron concentrations greater than ½HRL, or >0.7 mg/L, were found in 20 of 228 ground water samples (8.8%) and no surface water samples (0%). Boron concentrations greater than the HRL, or >1.4 mg/L, were found in seven of 228 ground water samples (3.1%) and no surface water samples (0%). The seven HRL exceedances were found at five systems. The highest concentration detected in ground water was approximately 3.32 mg/L, and the highest concentration in surface water was 0.345 mg/L (Seidel, 2006). The median concentrations were 0.0514 mg/L in ground water and 0.029 mg/L in surface water (Frey *et al.*, 2004).

Although the survey was not statistically representative, it indicates some general trends. On the whole, boron contamination of surface water is less significant than contamination of ground water. No geographic trends were evident in ground water results, but surface water contamination appeared to be more prevalent in the Western U.S. than the Eastern U.S. Longitudinal sampling (i.e., sampling at multiple points along the path of water undergoing treatment) at 15 systems revealed that a wide variety of treatment techniques were largely ineffective at removing boron, so boron concentrations in source water (such as those collected in this study) are likely to be indicative of concentrations in finished water (Frey *et al.*, 2004).

Community Water System Survey (CWSS)

The 2000 Community Water System Survey (CWSS) (USEPA, 2002a; 2002b) gathered data on the financial and operating characteristics of a random sample of community water systems nationwide. In addition, the Survey asked all "very large" community water systems, those that serve more than 500,000 people (a total of 83 systems), to provide monitoring results for five regulated compounds (arsenic, atrazine, 2,4-D, simazine, and glyphosate) and four unregulated compounds (radon, methyl tertiary-butyl ether [MTBE], metolachlor, and boron), including results from raw water at each intake and from finished water at each treatment plant. EPA received completed questionnaires from 58 systems. However, not all systems answered every question. Note that because reported results are incomplete, they are more illustrative than statistically representative.

Results of raw water monitoring are aggregated by type of intake. In raw ground water, 34 observations of boron occurrence were reported. Among detects, the median concentration was 120 µg/L and the 90th percentile concentration was 273 µg/L. Non-detects were reported at 2.6 percent of ground water intakes. In raw surface water, 15 observations of boron occurrence were reported. Among detects, the median concentration was 59 µg/L and the 90th percentile

concentration was 180 µg/L. Non-detects were reported at 22.2 percent of surface water intakes (USEPA, 2002b).

Results of finished water monitoring are aggregated by system type. At systems primarily served by ground water, 5 observations of boron occurrence were reported. Among detects, the median concentration was 102 µg/L and the 90th percentile concentration was 234 µg/L. No non-detects were reported. At systems primarily served by surface water, 14 observations of boron occurrence were reported. Among detects, the median concentration was 56 µg/L and the 90th percentile concentration was 500 µg/L. Non-detects were reported at 9.1 percent of treatment plants. At systems primarily served by purchased water, 6 observations of boron occurrence were reported. Among detects, the median concentration was 164 µg/L and the 90th percentile concentration was 200 µg/L. Non-detects were reported at 1.8 percent of treatment plants (USEPA, 2002b).

3.4 Technology Assessment

3.4.1 Analytical Methods

Boron can be detected using EPA Method 200.7. Method 200.7 relies on inductively coupled plasma-atomic emission spectrometry (ICP-AES). A full description of EPA Method 200.7 can be found in EPA's *Methods for the Determination of Metals in Environmental Samples Supplement 1* (USEPA, 1994b). A brief summary of this method is provided below. It should be noted that the analytical result of this method is for the amount of elemental boron; the method does not identify the boron compound(s) present.

EPA Method 200.7

In EPA Method 200.7 (Revision 4.4), "*Determination of Metals and Trace Elements in Water and Wastes by ICP/Atomic Emission Spectrometry,*" an aliquot of a well-mixed, acid-preserved aqueous sample is accurately transferred for sample processing. The sample is made up to one-half the original aliquot volume, mixed, and then allowed to settle overnight if the prepared aliquot contains undissolved material. Note that in low-turbidity water, boron determinations can be completed by "direct analysis" of acid-preserved samples. The analysis involves multielemental determinations by ICP-AES using sequential or simultaneous instruments. The instruments measure characteristic atomic-line emission spectra by optical spectrometry. Samples are nebulized and the resulting aerosol is transported to the plasma torch. Element-specific emission spectra are produced by a radio-frequency ICP. The spectra are dispersed by a grating spectrometer, and the intensities of the line spectra are monitored at specific wavelengths by a photosensitive device (USEPA, 1994b).

Note that boron samples can become contaminated by borosilicate glass. Only plastic or polytetrafluoroethylene (PTFE) labware should be used when collecting, storing, and handling water samples for boron analysis (USEPA, 1994b).

The method detection limit (MDL[1]) for boron using Method 200.7 is reported to be 0.003 mg/L (USEPA, 1994b). The average recovery ranges from 97 to 98 percent depending on the spike concentration and whether tap or well water was used.

Another possible method for boron detection is Standard Method (SM) 4500-B B. The analytical range for this method is between 100 to1,000 µg/L. This method, known as the Curcumin Method, is available in the 19th edition of *Standard Methods for the Examination of Water and Wastewater* (AWWA, 1995).

3.4.2 Treatment Technologies

Treatment technologies do not influence the determination of whether or not a contaminant should be regulated. However, before a contaminant can be regulated with a national primary drinking water regulation (NPDWR), treatment technologies must be readily available. There is no evidence that boron and boron compounds are significantly removed by conventional treatments, such as coagulation/flocculation, sedimentation, and inert media filtration. Two treatment technologies that may be appropriate are ion exchange and reverse osmosis.

Ion exchange involves the selective removal of charged inorganic species from water using an ion-specific resin. The surface of the ion exchange resin contains charged functional groups that hold ionic species by electrostatic attraction. As water passes by the resin, charged ions on the resin surface are exchanged for the contaminant species in the water. When all of the resin's available exchange sites have been replaced with ions from the feed water, the resin is exhausted and must be regenerated or replaced.

Wong (1984) evaluated eight technologies for their ability to remove boron from evaporator product water at power plants. Boron concentration in the evaporator-product water averaged 11 mg/L, and ranged as high as 38 mg/L. Only three technologies successfully reduced boron levels to below 0.3 mg/L. These were a boron-specific ion exchange resin, a process of coagulation, precipitation and filtration, and a strong-base anion-exchange resin. Wong dismissed the coagulation, filtration, and filtration process as unacceptable due to high chemical dosage requirements and high operating cost. Of the two ion exchange methods, Wong determined that the strong-base anion exchange resin would have lower regeneration costs, at least in the case of the evaporator product water, which is low in dissolved solids.

Reverse osmosis (RO) is similar to other membrane processes, such as ultrafiltration and nanofiltration, in that water passes through a semi-permeable membrane. However, in the case of RO, the membrane is non-porous. RO involves the use of applied hydraulic pressure to oppose the osmotic pressure across the membrane, forcing the water from the concentrated-

[1] The Method Detection Limit is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon the precision of a series of replicate measurements of the analyte at low concentrations. The MDL incorporates estimates of the accuracy of the determination. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

solution side to the dilute-solution side. The water dissolves into the membrane, diffuses across, then dissolves out into the permeate. Most inorganic and many organic contaminants are rejected by the membrane and will be retained in the concentrate.

Folster *et al.* (1980) tested hollow-fiber (HF) RO and spiral-wound (SW) RO in two separate treatment plants in New Mexico. At the treatment plant in San Jon, with influent boron levels of 0.75 mg/L, HF RO and SW RO removed 15 percent and 3 percent of boron, respectively. At Alamogordo, however, where influent concentrations were lower (0.09 mg/L), HF RO and SW RO were ineffective; in fact, boron concentrations rose to 0.14 mg/L and 0.13 mg/L, respectively. These findings suggest that the potential for RO use in boron treatment is limited.

3.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate boron with a national primary drinking water regulation (NPDWR). While boron was found at levels greater than the HRL (and ½ the HRL) in several of the ground water systems surveyed by NIRS, it was not found at levels greater than the HRL (or ½ the HRL) in the surface waters sources evaluated in the AWWARF study. Taking this surface water information into account, the Agency believes that the overall national occurrence and exposure from both surface and ground water systems together is likely to be lower than the values observed for the NIRS ground water data. Because boron is not likely to occur at levels of concern when considering both surface and ground waters systems, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

The Agency encourages those States with public water systems that have boron at concentrations above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory for boron to provide more recent health information. The updated Health Advisory will provide information to any States with public water systems that may have boron above the HRL.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

3.6 References

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. *Toxicological Profile for Boron*. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services. Available on the Internet at: http://www.atsdr.cdc.gov/toxprofiles/tp26.html.

Allen, B.C., P.L. Strong, C.J. Price, S.A. Hubbard, and G.P. Datson. 1996. Benchmark Dose Analysis of Developmental Toxicity in Rats Exposed to Boric Acid. *Fundamental and Applied Toxicology.* Vol. 32. pp. 194-204. (As cited in EPA, 2004b).

American Water Works Association (AWWA). 1995. *Standard Methods for the Examination of Water and Wastewater, 19th Edition.*

Biggar, J.W. and M. Fireman. 1960. Boron adsorption and release by soils. *Soil Science Society of America Proceedings*. 24:115-120. (As cited in ATSDR, 1992).

Bingham, F.T., A.L. Page, N.T. Coleman, and K. Flach. 1971. Boron adsorption characteristics of selected amorphous soils from Mexico and Hawaii. *Soil Science Society of America Proceedings*. 35:546-550. (As cited in ATSDR, 1992).

Bonn, B.A. 1999. *Selected Elements and Organic Chemicals in Bed Sediment and Fish Tissue of the Tualatin River Basin, Oregon, 1992-96*. U.S. Geological Survey Water-Resources Investigations Report 99-4107, 61 p. Available on the Internet at: http://or.water.usgs.gov/pubs_dir/Pdf/99-4107.pdf.

Budavari, S. (ed.) 1989. *The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals*. Rahway, NJ: Merck and Co., Inc. (As cited in HSDB, 2004).

Butterwick, L., N. de Oude, and K. Raymond. 1989. Safety Assessment of Boron in Aquatic and Terrestrial Environments. *Ecotoxicology and Environmental Safety*. 17:339-371. (As cited in ATSDR, 1992).

Clark, G.M. and T.R. Maret. 1998. *Organochlorine compounds and trace elements in fish tissue and bed sediments in the lower Snake River Basin, Idaho and Oregon*. U.S. Geological Survey Water-Resources Investigations Report 98-4103, 35 p. Available on the Internet at: http://id.water.usgs.gov/PDF/wri984103/ORGANOSX.PDF.

Dawson, B.J.M. 2001. *Ground-water quality in the southeastern Sacramento Valley aquifer, California*. U.S. Geological Survey Water-Resources Investigations Report 01-4125, 24 p. Available on the Internet at: http://pubs.usgs.gov/wri/wri014125/wrir01-4125.pdf.

Field, E.A., C.J. Price, M.C. Marr, C.B. Myers, R.E. Morrissey, and B.A. Schwetz. 1989. *Final report on the developmental toxicity of boric acid (CAS No. 10043-35-3) in CD-1-Swiss Mice.* NTP Final Report No. 89-250. (As cited in USEPA, 2004b).

Folster, H.G., D.B. Wilson, S. Hanson, and R. Duran. 1980. New Mexico Water Resources Research Institute. *Water treatment for small public supplies*. WRRI Report No. 119.

Frey, M.M., C. Seidel, M. Edwards, J. Parks, and L. McNeill. 2004. *Occurrence Survey for Boron and Hexavalent Chromium.* AWWARF Report 91044F.

Graedel, T.E. 1978. Inorganic elements, hydrides, oxides, and carbonates. In: *Chemical compounds in the atmosphere*. New York, NY: Academic Press, pp. 35-49. (As cited in ATSDR, 1992).

Hazardous Substances Data Bank (HSDB). 2004. Search for boron. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB, accessed November 1, 2004.

Heindel, J.J., C.J. Price, E.A. Field, M.C. Marr, C.B. Myers, R.E. Morrissey, and B.A. Schwetz. 1992. Developmental toxicity of boric acid in mice and rats. *Fundamental and Applied Toxicology*. Vol. 18. pp. 266-277. (As cited in USEPA, 2004b).

Heindel, J.J., C.J. Price, and B.A. Schwetz. 1994. The developmental toxicity of boric acid in mice, rats and rabbits. *Environmental Health Perspectives Supplements*. Vol. 102, No. S7. pp. 107-112. (As cited in USEPA, 2004b).

Institute of Medicine (IOM). 2001. Boron. Chapter in *Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium and zinc.* Washington, DC: National Academy Press. p. 510-521.

Iyengar, G.V., W.B. Clarke, R.G. Downing, and J.T. Tanner. 1988. Lithium in biological and dietary materials. In: *Proceedings of the 5th International Workshop on Trace Element Analytical Chemistry in Medicine and Biology*. Vol. 5. pp. 267-269. (As cited in USEPA, 2004a).

Keren, R. and U. Mezuman. 1981. Boron adsorption by clay minerals using a phenomenological equation. *Clays and Clay Minerals*. 29:19-204. (As cited in ATSDR, 1992).

Keren, R., R.G. Gast, and B. Bar-Yosef. 1981. pH-dependent boron adsorption by Na-montmorillonite. *Soil Science Society of America Journal.* 45:45-48. (As cited in ATSDR, 1992).

Moore, J.W. 1991. *Inorganic Contaminants of Surface Water: Research and Monitoring Priorities.* New York, NY: Springer-Verlag.

Minnesota Pollution Control Agency (MPCA). 1998. *Baseline Water Quality of Minnesota's Principal Aquifers*. Available on the Internet at: http://www.pca.state.mn.us/water/groundwater/gwmap/gwbaselinerpt.html. Accessed October 13, 2004. Last updated October 28, 1998.

Parks, W.L. and J.L. White. 1952. Boron retention by clay and humus systems saturated with various cations. *Soil Science Society of America Proceedings*. pp. 298-300. (As cited in ATSDR, 1992).

Peterson, D.A. and R.B. Zelt. 1999. *Element concentrations in bed sediment of the Yellowstone River Basin, Montana, North Dakota, and Wyoming--A retrospective analysis.* U.S. Geological Survey Water-Resources Investigations Report 99-4185, 23 p. Available on the Internet at: http://water.usgs.gov/pubs/wri/wri994185/pdf/wri994185.pdf.

Price, C.J., E.A. Field, M.C. Marr, C.B. Myers, R.E. Morrissey, and B.A. Schwetz. 1990. *Developmental toxicity of boric acid (CAS No. 10043-35-3) in Sprague Dawley rats.* NTP Report No. 90-105. (As cited in USEPA, 2004b).

Price, C.J., M.C. Marr, and C.B. Myers. 1994. *Determination of the no-observable-adverse-effect level (NOAEL) for developmental toxicity in Sprague-Dawley (CD) rats exposed to boric acid in feed on gestational days 0 to 20, and evaluation of postnatal recovery through postnatal day 21*. Final report. (As cited in USEPA, 2004b).

Price, C.J., P.L. Strong, M.C. Marr, C.B. Myers, and F.J. Murray. 1996. Developmental toxicity NOAEL and postnatal recovery in rats fed boric acid during gestation. *Fundamental and Applied Toxicology*. Vol. 32. pp. 179-193. (As cited in USEPA, 2004b).

Rai, D., J.M. Zachara, A.P. Schwab, R. Schmidt, D. Girvin, and D. Rogers. 1986. Chemical attenuation rates, coefficients, and constants in leachate migration. Vol. 1. A critical review. Report to Electric Power Research Institute, Palo Alto, CA by Battelle, Pacific Northwest Laboratories, Richland, WA. Research Project 2198-1. (As cited in ATSDR, 1992).

Rio Tinto Borax. 2004. Rio Tinto Borax 2003 Report on Sustainability: Company Overview, Borax Profile. Available on the Internet at: http://www.borax.com/sereport03. Accessed December 30, 2004.

Sakata, M. 1987. Relationship between adsorption of arsenic (III) and boron by soil and soil properties. *Environmental Science and Technology*. 21:1126-1130. (As cited in ATSDR, 1992).

Seidel, C. 2006. Email Communication to Brent Ranalli at The Cadmus Group, Inc. [concerning boron data from an AWWARF-sponsored study, with data in an attached spreadsheet]. Denver, CO: McGuire Malcolm Pirnie; May 19, 2006.

United States Environmental Protection Agency (USEPA). 1987. Toxic air pollutant/source crosswalk: A screening tool for locating possible sources emitting toxic air pollutants. Office of Air Quality and Planning Standards. EPA Report 450/4-87-023a. (As cited in ATSDR, 1992).

USEPA. 1994a. *Reregistration Eligibility Decision (RED) - Boric Acid and its Sodium Salts*. Office of Prevention, Pesticides, and Toxic Substances. Available on the Internet at: http://www.epa.gov/oppsrrd1/REDs/old_reds/boric_acid_salts.pdf.

USEPA. 1994b. Method 200.7: Determination of Metals and Trace Elements in Water and Wastes by Inductively Coupled Plasma-Atomic Emission Spectrometry Revision 4.4. Martin, T.D., C.A. Brockhoff, J.T. Creed, and EMMC Methods Work Group. In: *Methods for the Determination of Metals in Environmental Samples Supplement 1*. EPA Report 600-R-94-111.

USEPA. 2002a. *Community Water System Survey 2000. Volume I: Overview*. EPA Report 815-R-02-005A. December 2002. Available on the Internet at: http://www.epa.gov/safewater/consumer/pdf/cwss_2000_volume_i.pdf.

USEPA. 2002b. *Community Water System Survey 2000. Volume II: Detailed Tables and Survey Methodology*. EPA Report 815-R-02-005B. Available on the Internet at: http://www.epa.gov/safewater/consumer/pdf/cwss_2000_volume_ii.pdf.

USEPA. 2004a. Integrated Risk Information System (IRIS), Boron and Compounds. August 2004. Available on the Internet at: http://www.epa.gov/iris/subst/0410.htm. Accessed February 2, 2005.

USEPA. 2004b. *Toxicological Review of Boron and Compounds.* EPA Report 635-04-052. June 2004. Available online at: http://www.epa.gov/iris/toxreviews/0410-tr.pdf.

USEPA. 2006a. TRI Explorer: Trends. Searches for boron trichloride, boron trifluoride. Available on the Internet at: http://www.epa.gov/triexplorer/trends.htm, last modified June 8, 2005, accessed February 8, 2006.

USEPA. 2006b. *The Analysis of Occurrence Data from the Unregulated Contaminant Monitoring (UCM) Program and National Inorganics and Radionuclides Survey (NIRS) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-009. Draft. December 2006.

United States Geological Survey (USGS). 2004. Mineral Commodity Summaries, January 2004--Boron. January 2004. Available on the Internet at: http://minerals.usgs.gov/minerals/pubs/commodity/boron/boronmcs04.pdf.

Waggot, A. 1969. An investigation of the potential problem of increasing boron concentrations in rivers and water courses. *Water Research*. 3:749-765. (As cited in ATSDR, 1992).

Warner, K.L. 1999. *Analysis of nutrients, selected inorganic constituents, and trace elements in water from Illinois community-supply wells, 1984-91*. U.S. Geological Survey Water-Resources Investigations Report 99-4152, 47 p. Available on the Internet at: http://il.water.usgs.gov/proj/lirb/pubs/pdfs/topicalbook.pdf.

Weast, R.C. (ed.). 1988. *CRC Handbook of Chemistry and Physics*. 68th ed. Boca Raton, FL: CRC Press Inc. (As cited in ATSDR, 1992 and HSDB, 2004).

Windholz, M., S. Budavari, R.F. Blumetti, E.S. Otterbein (eds.) 1983. *The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals*, 10th Edition. Rahway, NJ: Merck and Co., Inc.

Wong, J.M. 1984. Boron Control in Power Plant Reclaimed Water for Potable Reuse. *Environmental Progress*. 3(1):5-11.

Chapter 4:
DCPA Mono- and Di-Acid Degradates

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

Dimethyl tetrachloroterephthalate (DCPA), a synthetic organic compound (SOC) marketed under the trade name "Dacthal," is a pre-emergent herbicide historically used to control weeds in ornamental turf and plants, strawberries, seeded and transplanted vegetables, cotton, and field beans. As of 1990, more than 80 percent of its use was for turf, including golf courses and home lawns. Available data indicate that DCPA use declined significantly over the course of the 1990s. On July 27, 2005, in response to concerns about groundwater contamination (especially for one of the DCPA degradates), the registrant voluntarily terminated most uses for products containing DCPA. DCPA is currently registered only for use on sweet potatoes, eggplant, kale, and turnips.

DCPA is not particularly mobile or persistent in the environment. Biodegradation and volatilization are the primary dissipation routes. Degradation of DCPA forms two breakdown products, the mono-acid degradate (monomethyl tetrachloroterephthalate or MTP) and the di-acid degradate (tetrachloroterephthalic acid or TPA). The di-acid, which is the major degradate, is mobile and persistent in the field, with a potential to leach into water.

The present toxicity database for MTP and TPA is not sufficient to derive reference doses (RfDs) for these two chemicals. However, since the available data indicate that neither MTP nor TPA is more toxic than their parent compound, DCPA, the Agency believes that the RfD for the DCPA parent would be protective against exposure from the two DCPA metabolites. Both compounds are formed in the body from the DCPA parent, and therefore the toxicity of the degradates is reflected in the observed toxicity of the parent compound. The RfD of 0.01 mg/kg/day for DCPA is based on a chronic rat study with a no-observed-adverse-effect level (NOAEL) of 1.0 mg/kg/day, and incorporates an uncertainty factor of 100. Using the DCPA RfD of 0.01 mg/kg/day and a 20 percent screening relative source contribution (RSC), the Agency calculated a health reference level (HRL) of 0.07 mg/L or 70 µg/L for DCPA and used this HRL for TPA and MTP. No sensitive subpopulations have been identified. Based on the cancer data for DCPA and evidence that neither TPA nor DCPA is mutagenic, the Agency concludes that TPA is unlikely to pose a cancer risk.

EPA evaluated the occurrence of the DCPA degradates in drinking water based on data from the first Unregulated Contaminant Monitoring Regulation (UCMR 1). The results for the two degradates are reported in aggregate. While the UCMR 1 data indicate that the DCPA degradates were the most commonly reported analytes in the monitoring survey (detected at a minimum reporting level or MRL of 1 µg/L in 772 samples from 175 of the 3,868 public water systems or PWSs sampled, in 24 States and 1 Territory), very few systems exceeded thresholds of health concern. Approximately 0.05 percent of the 3,868 PWSs sampled had a detection of the DCPA degradates at levels greater than ½ the DCPA HRL (35 µg/L), affecting approximately 0.33 percent of the population served. Approximately 0.03 percent of the 3,868 PWSs sampled had a detection of the DCPA degradates at levels greater than the DCPA HRL (70 µg/L), affecting less than 0.01 percent of the population served.

EPA evaluated additional data on the occurrence of DCPA and its degradates in ambient and drinking water from several sources. These supplemental sources included: the United

States Geological Survey's (USGS's) National Ambient Water Quality Assessment (NAWQA) program, studies performed by the DCPA or dacthal registrant, the Pesticides in Ground Water Database, and the National Pesticide Survey.

The Agency has made a preliminary determination not to regulate the DCPA mono-acid degradate and/or the DCPA di-acid degradate with a national primary drinking water regulation (NPDWR). Because these degradates appear to occur infrequently at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

The Agency encourages those States with public water systems that have detects for these degradates to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory for the DCPA parent to include the mono- and di-acid degradates, as well as any recent health information related to these compounds. The updated Health Advisory will provide information to any States with public water systems that may have DCPA degradates at levels above the HRL.

The Agency's preliminary regulatory determination for these contaminants is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

a.i.	Active Ingredient
AOAC	Association of Official Analytical Chemists
AOP	Advanced Oxidation Process
APHA	American Public Health Association
ASTM	American Society for Testing and Materials
CAS	Chemical Abstracts Service
CCL 2	Second Contaminant Candidate List
CWS	Community Water System
DCPA	Dimethyl tetrachloroterephthalate (Dacthal)
GAC	Granular Activated Carbon
GC/ECD	Gas Chromatography with Electron Capture Detection
HRL	Health Reference Level
MDL	Method Detection Limit
MF	Microfiltration
MRL	Minimum Reporting Level
MTBE	Methyl tertiary-butyl ether
MTP	Monomethyl tetrachloroterephthalate
NAWQA	National Water Quality Assessment
NCFAP	National Center for Food and Agricultural Policy
NF	Nanofiltration
NOAEL	No Observed Adverse Effect Level
NPDWR	National Primary Drinking Water Regulation
NPS	National Pesticide Survey
NTNCWS	Non-Transient Non-Community Water System
PGWDB	Pesticides in Ground Water Database
PWS	Public Water System
RED	Reregistration Eligibility Decision
RfD	Reference Dose
RL	Reporting Limit
RO	Reverse Osmosis
RSC	Relative Source Contribution
SOC	Synthetic Organic Compound
TPA	Tetrachloroterephthalic Acid
UCMR 1	First Unregulated Contaminant Monitoring Regulation
UF	Ultrafiltration or Uncertainty Factor
USGS	United States Geological Survey
UV	Ultraviolet

4 DCPA Mono- and Di-Acid Degradates

4.1 Definition

Dimethyl tetrachloroterephthalate (DCPA), a synthetic organic compound (SOC), is a phthalate herbicide. Common names for DCPA include Dacthal, 2,3,5,6-tetrachloro-1,4-benzenedicarboxylic acid dimethyl ester, dimethyl 2,3,5,6-tetrachloroterephthalate, chlorthal dimethyl, and Rid. The Chemical Abstracts Service (CAS) registry number for DCPA is 1861-32-1. In the environment, DCPA is readily metabolized. Tetrachloroterephthalic acid (TPA or di-acid; $C_8H_2Cl_4O_4$; CAS number 2136-79-0) is the only significant DCPA metabolite, with monomethyl tetrachloroterephthalic acid (mono-acid; $C_9H_4Cl_4O_4$; CAS number 887-54-7) as a minor metabolite (USEPA, 1998).

4.1.1 Properties and Sources

DCPA is synthesized for use as a pre-emergent herbicide on annual grasses and broadleaf weed species. As of 1990, more than 80 percent of DCPA use was for turf, including golf courses and home lawns (USEPA, 1990a). Extoxnet (1996) estimated that roughly 20 percent was used around homes and gardens (Extoxnet, 1996). DCPA does not occur naturally in the environment. As of 1998, the manufacturing of DCPA was limited to three products registered to ISK Biosciences Corporation (formerly Fermenta ASC Corporation) (USEPA, 1998). In 2005, many uses of DCPA were voluntarily cancelled by the current registrant, AMVAC (70 FR 43408). Technical grade DCPA exists as a colorless or white crystal. DCPA is virtually insoluble in water. It is soluble in the following organic solvents in descending order of solubility: benzene, toluene, xylene, dioxane, acetone, and carbon tetrachloride (USEPA, 1998). Biodegradation is the primary dissipation process of DCPA, and degradation can occur by several lytic and metabolic pathways (e.g., photolysis, or aerobic and anaerobic metabolism). With the hydrolyzation of one ester bond, DCPA degrades to the mono-acid. When the second ester bond is hydrolyzed, it is degraded to the di-acid. Some physical and chemical properties of DCPA are listed in Exhibit 4-1. No information is available on the physical and chemical properties of the mono- and di-acid degradates.

Exhibit 4-1: Physical and Chemical Properties of DCPA

Identification	
CAS Number	1861-32-1
Molecular Formula	$C_{10}H_6Cl_4O_4$
Physical and Chemical Properties	
Boiling Point	360 - 370 °C [1]
Melting Point	156 °C [2]
Molecular Weight	331.99 g/mol [1]
Log K_{oc}	3.81 [3]
Log K_{ow}	4.19 [4]
Water Solubility	0.5 mg/L at 25 °C [5]
Vapor Pressure	2.5 x 10^{-6} mm Hg at 25 °C [6]
Henry's Law Constant	2.18 x 10^{-6} atm-m^3/mol [7]
Freundlich Isotherm Constant (K)	--

[1] *Tomlin, 1997 (as cited in HSDB, 2004)*

[2] *Budavari, 1989 (as cited in HSDB, 2004)*

[3] *Lyman et al., 1990 (as cited in HSDB, 2004)*

[4] *Hansch et al., 1995 (as cited in HSDB, 2004)*

[5] *Yalkowsky and Dannenfelser, 1992 (as cited in HSDB, 2004)*

[6] *Glotfelty et al., 1984 (as cited in HSDB, 2004)*

[7] *HSDB, 2004*

4.1.2 Environmental Fate and Behavior

DCPA is neither particularly persistent nor mobile in the environment. Although DCPA is relatively stable to hydrolysis and photolysis, it is susceptible to microbial degradation and volatilization. In laboratory studies, DCPA half-lives ranged between 15 and 30 days (USEPA, 1998). In most soils and ground water, the half-life of DCPA is more variable, ranging from 14 to 100 days (Wauchope *et al*., 1992 as cited in Extoxnet, 1996). DCPA's adsorption to clay and organic matter results in minimal soil mobility (HSDB, 2004).

Microbial degradation of DCPA occurs under aerobic and anaerobic soil conditions. The degradation rate increases with temperature and soil moisture (USEPA, 1998). The primary product of both aerobic and anaerobic metabolism is the di-acid degradate, which is mobile, persistent, and will leach in any type of soil. Under anaerobic soil conditions, the estimated half-life of DCPA is 37 to 59 days, with the final product being the di-acid degradate (USEPA, 1998). Under aerobic conditions, the estimated half-life is 18 to 37 days. In a 1993 study by Wettasinghe and Tinsley cited in the Reregistration Eligibility Decision (RED), essentially all of

the DCPA was transformed to di-acid DCPA after 197 days, although minor amounts of mono-acid DCPA were observed (USEPA, 1998). In a 300-day study, the half-life of mono-acid DCPA was about 2.8 days, while di-acid DCPA was persistent and barely degraded at all (Doran, 1990 as cited in USEPA, 1998).

Accumulation of DCPA in water is a major fate process for this contaminant. There is virtually no degradation of DCPA in water ranging from moderately acidic to moderately alkaline (pH 5.0 to pH 9.0). Breakdown is due to the action of sunlight and the half-life is greater than 1 week (Extoxnet, 1996). Substantial bioaccumulation of DCPA was observed in bluegill sunfish. Bioconcentration factors of 1,984, 777, and 2,574 were measured in whole fish, edible tissue, and viscera, respectively (USEPA, 1998). DCPA metabolism in fish tissues is inconsequential, but demethylation is detectable (Szalkowski *et al*., 1980; Szalkowski *et al*., 1981 both as cited in USEPA, 1998).

Volatilization is also a major route of DCPA dissipation from soil. Despite a relatively low Henry's Law Constant (see Exhibit 4-1) and a high log K_{oc}, numerous published studies document the volatility of parent DCPA (USEPA, 1998). Nash and Gish (1989 as cited in USEPA, 1998) suggest that DCPA volatilization may be controlled by adsorption and diffusion; thus vapor pressure would not be a good indicator (USEPA, 1998). Volatilization accounts for 20 to 40 percent of DCPA loss under normal soil conditions, but can be significantly higher with increased field moisture and soil temperature (USEPA, 1998).

In the atmosphere, DCPA exists in both the vapor phase and the particulate phase. In the vapor phase, DCPA degrades through a reaction with photochemically produced hydroxyl radicals and has an estimated half-life of 36 days (HSDB, 2004). Wet and dry deposition can physically remove particulate-phase DCPA from the atmosphere (HSDB, 2004). Atmospheric transport has been implicated in DCPA contamination of crops that were not treated with DCPA (USEPA, 1998).

4.2 Health Effects

Currently, no subchronic or chronic studies are available to assess the toxicological effects of MTP (the mono-acid degradate) and 3 studies in rats (30 and 90-day feeding studies and a one-generation reproductive study) are available for TPA (the di-acid degradate). The effects of exposure were mild (weight loss and diarrhea) and occurred at doses greater than or equal to 2,000 mg/kg/day. No reproductive effects were observed.

The present toxicity database for MTP and TPA is not sufficient to derive reference doses (RfDs) for these two chemicals. However, since the available data indicate that neither MTP nor TPA are more toxic than their parent compound, DCPA, the Agency suggests that the RfD for the DCPA parent would be protective against exposure from these two DCPA metabolites (USEPA, 1998). Both compounds are formed in the body from the DCPA parent and therefore, the toxicity of these degradates is reflected in the toxicity of the parent. The RfD for DCPA is 0.01 mg/kg/day based on a chronic rat study (ISK Biotech Corporation, 1993) with a "no-observed-adverse-effect level" (NOAEL) of 1.0 mg/kg/day and an uncertainty factor of 100 for rat to human extrapolation and intra-species variability.

No carcinogenicity studies have been performed using either TPA or MTP. Based on the cancer data for the parent and lack of mutagenicity for TPA and DCPA, the Agency (USEPA, 2004) concludes that TPA is unlikely to pose a cancer risk. Klopman *et al.* (1996) evaluated the carcinogenic potential of TPA based on its chemical and biological properties, as well as by a variety of computational tools, and determined that it did not present any substantial carcinogenic risk. There was suggestive evidence that DCPA could be carcinogenic based on an increased incidence of thyroid and liver tumors in rats. The presence of hexachlorobenzene and dioxin as impurities in the material tested could have contributed to the cancer risk.

Using the DCPA RfD of 0.01 mg/kg/day (USEPA, 1994) and a 20 percent screening relative source contribution (RSC), the Agency calculated a health reference level (HRL) of 0.07 mg/L or 70 µg/L for DCPA and used this HRL for TPA and MTP.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. There are no data that identify a particular sensitive population for DCPA exposure. Results of a single developmental study indicate that exposure to pregnant dams with doses less than or equal to 2,500 mg/kg/day of TPA via gavage did not have an adverse effect on the fetus. EPA did not identify any data that suggest gender-related differences in toxicity or sensitivity in the elderly.

4.3 Occurrence and Exposure

4.3.1 Use and Environmental Release

DCPA, marketed under the trade name "Dacthal," was first introduced as a pesticide in 1958. Until recently it was registered for both commercial and residential use, including use as a selective pre-emergence weed control on ornamental turf and plants, strawberries, seeded and transplanted vegetables, cotton, and field beans (USEPA, 1998). On July 27, 2005, in response to concerns about groundwater contamination (especially for one of the DCPA degradates), the registrant voluntarily terminated residential uses of DCPA and many uses on vegetable and nut products (70 FR 43408). The only uses retained were for sweet potatoes, eggplant, kale, and turnip.

To prevent the direct ingestion of DCPA by humans or livestock, DCPA use is subject to certain restrictions. These restrictions prohibit: direct application of DCPA to water or wetlands; effluent discharge containing DCPA to sewage plants without notifying the proper authorities; effluent discharge to rivers, lakes, ponds or other bodies of fresh- or saltwater; feeding clippings or vegetation treated with DCPA to livestock; and grazing livestock in treated areas (USEPA, 1998).

Several studies and reports, described below, provide estimates of recent DCPA use in the United States. However, these estimates should be interpreted with caution, because none of them consider all uses. In 1990, an EPA report suggested that more than 80 percent of DCPA use was on turf, including golf courses and home lawns (USEPA, 1990a). It is not clear how the percentage of turf use may have changed since then, and what percentage of DCPA use (including turf use) is commercial as opposed to residential.

EPA's estimate of commercial DCPA use in the early 1990s, based on proprietary data as well as data from the United States Department of Agriculture, the State of California, and the National Center for Food and Agricultural Policy (NCFAP), is that approximately 1.6 million pounds of active ingredient (a.i.) (range of 1.1 million pounds to 2.1 million pounds) were used annually on about 241,000 to 404,000 acres (USEPA 1998). That estimate includes application on golf courses and sod farms (totaling approximately 225,000 to 450,000 pounds a.i. on 37,000 to 58,000 acres), but does not include residential uses. Separately, EPA has estimated that annual use of DCPA in homes and gardens between 1994 and 1999 has consistently fallen in the range of 1 million to 3 million pounds a.i. (USEPA 1997, 1999, 2002)

NCFAP lists annual DCPA use on 26 crops totaling approximately 1.7 million pounds a.i. on approximately 273,000 acres around the year 1992, and annual DCPA use on 18 crops totaling approximately 0.6 million pounds a.i. on approximately 106,000 acres around the year 1997 (NCFAP, 2003). The NCFAP estimates are based on State-level usage patterns for the periods 1990-1993 and 1995-1998, keyed to State-level crop acreage for 1992 and 1997. Only cropland uses are included in these data; the estimates include sod farms but exclude golf courses and residential uses. For more information on NCFAP pesticide use estimates, see Chapter 2.

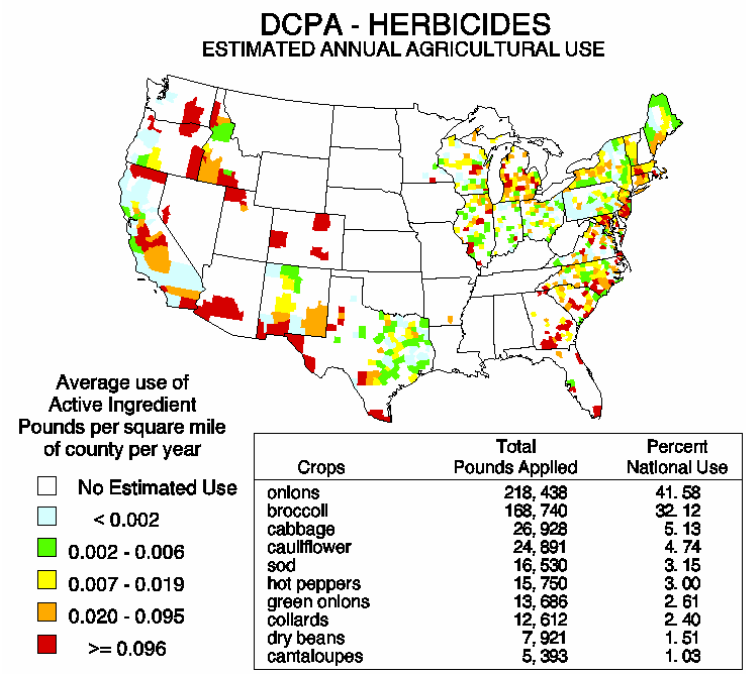
The United States Geological Survey (USGS) has combined State-level NCFAP pesticide usage data with more precise county-level Census of Agriculture acreage data to calculate national pesticide use (Thelin and Gianessi, 2000). Annual DCPA use around 1992 was estimated to be approximately 998,000 pounds a.i. on approximately 185,000 acres. While USGS has not published national estimates for 1997, an estimate of approximately 525,000 pounds a.i. can be inferred from the "total pounds applied" and "percent national use" data in the 1997 geographical distribution map (Exhibit 4-2). USGS estimates, based partly on NCFAP data, also have limitations; the estimates reflect cropland uses, including sod farming, but no other uses.

Total annual DCPA usage in recent years, including agricultural usage, commercial non-agricultural turf usage (e.g., on golf courses), and residential usage, can not be estimated with precision from the data summarized above. The annual total might fall anywhere between 1.5 million and 5 million pounds. No trends are apparent in the EPA estimates of homeowner use, but both the NCFAP data and the USGS estimates suggest a significant decline in agricultural use of DCPA during the 1990s (i.e., from 1.0-1.7 million pounds a.i. per year to 0.5-0.6 million pounds a.i. per year). With the recent termination of many DCPA uses, it is reasonable to expect that DCPA applications will continue to decline.

Exhibit 4-2 shows the estimated geographic distribution and intensity of typical annual DCPA use in the United States in the late 1990s. A breakdown of use by crop is also included. The map indicates that agricultural uses of DCPA are generally concentrated along the entire eastern seaboard, in the Great Lakes States, and in a large, ten-State area of the west, stretching from Washington and Idaho to California, Colorado, and Texas. The map was created by the USGS using NCFAP State-level estimates of pesticide use rates from 1995-1998 and county-level data on harvested crop acreage from the 1997 Census of Agriculture (USGS, 2004). Due to the nature of the data sources, non-agricultural uses are not reflected here and variations in use at the county-level are also not well represented (Thelin and Gianessi, 2000). For more

information on the USGS pesticide use maps, see Chapter 2. As noted above, approximately 80 percent of DCPA use is for weed control on turf, including golf courses, and home lawns; these uses are not represented in the map. The actual geographic distribution of most DCPA use, therefore, is not well known.

Exhibit 4-2: Estimated Annual Agricultural Use of DCPA (c. 1997)



Crops	Total Pounds Applied	Percent National Use
onions	218,438	41.58
broccoli	168,740	32.12
cabbage	26,928	5.13
cauliflower	24,891	4.74
sod	16,530	3.15
hot peppers	15,750	3.00
green onions	13,686	2.61
collards	12,612	2.40
dry beans	7,921	1.51
cantaloupes	5,393	1.03

Source: USGS, 2004

The DCPA mono- and di-acid degradates have no known uses, and the application of DCPA is the only known source of the degradates in the environment.

4.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are the source of most drinking water. Data on the occurrence of DCPA and its mono-acid degradate in ambient surface and ground water are available from the National Water Quality Assessment (NAWQA) program of the USGS. For details on the NAWQA program, see discussion in Chapter 2. NAWQA data have been analyzed independently by USGS and EPA. In addition, several smaller studies of ambient occurrence were conducted for purposes of pesticide reregistration.

NAWQA National Pesticide Synthesis

Surface Water and Ground Water

Under the NAWQA program, USGS monitored DCPA (listed as "dacthal") and DCPA mono-acid degradate (listed as "dacthal monoacid") between 1992 and 2001 in representative watersheds and aquifers across the country. Reporting limits varied but did not exceed 0.003 µg/L for DCPA and 0.070 µg/L for the degradate.

In surface water NAWQA samples (Exhibit 4-3), DCPA was found at frequencies ranging from 6.34% of samples in undeveloped areas to 11.46% of samples in agricultural settings, 15.4% of samples in mixed land use settings, and 21.78% of samples in urban areas. The higher frequency of occurrence in samples from urban areas may reflect that the majority of DCPA use is on turf (e.g., golf courses and lawns) rather than on agricultural crops. The 95th percentile concentrations were a non-detect in undeveloped settings, 0.003 µg/L in agricultural settings, 0.004 µg/L in mixed land use settings, and 0.007 µg/L in urban land use settings. The highest concentration, estimated at 40 µg/L, was found at an agricultural site (Martin *et al.*, 2003).

Exhibit 4-3: USGS National Synthesis Summary of NAWQA Monitoring of DCPA (Dacthal) in Ambient Surface Water, 1992-2001

Land Use Type	No. of Samples (and No. of Sites)	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,890 (78)	11.46%	<RL	0.003 µg/L	40 µg/L (E)
Mixed	1,020 (47)	15.40%	<RL	0.004 µg/L	0.179 µg/L
Undeveloped	60 (4)	6.34%	<RL	<RL	0.003 µg/L
Urban	902 (33)	21.78%	<RL	0.007 µg/L	0.045 µg/L

Abbreviations:

RL = Reporting limit. Reporting limits for dacthal varied, but did not exceed 0.003 µg/L.

E = Estimated (outside normal calibration limits)

Note: The USGS Pesticide National Synthesis used one year of data, generally the year with the most sampling results, to represent each site in this analysis. The sampling results were time-weighted, to eliminate bias from more frequent sampling at certain times of year. Detection Frequencies and Percentile Concentrations can be interpreted as representing annual occurrence. For instance, the detection frequency can be thought of as the percent of the year in which detections are found at a typical site in this land use category, and the 95th percentile concentration can be thought of as a concentration that is not exceeded for 95% of the year at a typical site in this land use category.

Source: Martin et al., 2003

The DCPA mono-acid degradate was not detected in surface water samples in undeveloped areas, mixed land use settings, or urban areas (Exhibit 4-4). It was detected in 0.18% of surface water samples in agricultural settings. The 95th percentile concentrations were

non-detects in all land use settings. The maximum surface water concentration in agricultural settings was 0.430 µg/L (Martin *et al.*, 2003).

Exhibit 4-4: USGS National Synthesis Summary of NAWQA Monitoring of DCPA's Mono-Acid Degradate in Ambient Surface Water, 1992-2001

Land Use Type	No. of Samples (and No. of Sites)	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,233 (48)	0.18%	<RL	<RL	0.430 µg/L
Mixed	561 (25)	0.00%	<RL	<RL	<RL
Undeveloped	19 (1)	0.00%	<RL	<RL	<RL
Urban	503 (18)	0.00%	<RL	<RL	<RL

Abbreviations:
RL = Reporting limit. Reporting limits for dacthal mono-acid varied, but did not exceed 0.070 µg/L.

Note: The USGS Pesticide National Synthesis used one year of data, generally the year with the most sampling results, to represent each site in this analysis. The sampling results were time-weighted to eliminate bias from more frequent sampling at certain times of year. Detection Frequencies and Percentile Concentrations can be interpreted as representing annual occurrence. For instance, the detection frequency can be thought of as the percent of the year in which detections are found at a typical site in this land use category, and the 95th percentile concentration can be thought of as a concentration that is not exceeded for 95% of the year at a typical site in this land use category.

Source: Martin et al., 2003

In ground water (Exhibit 4-5), DCPA detection frequencies ranged from 0% (no detects) in undeveloped settings to 0.44% in mixed land use (major aquifer) settings, 0.96% in urban settings, and 1.18% in agricultural settings. The 95th percentile concentrations were non-detects in all land use settings. The highest ground water concentration, estimated at 10 µg/L, was found at an agricultural site (Kolpin and Martin, 2003).

Exhibit 4-5: USGS National Synthesis Summary of NAWQA Monitoring of DCPA (Dacthal) in Ambient Ground Water, 1992-2001

Land Use Type	No. of Wells	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,443	1.18%	<RL	<RL	10 µg/L (E)
Mixed (Major Aquifer)	2,717	0.44%	<RL	<RL	0.004 µg/L
Undeveloped	67	0.00%	<RL	<RL	<RL
Urban	834	0.96%	<RL	<RL	0.011 µg/L

Abbreviations:
RL = Reporting limit. Reporting limits for dacthal varied, but did not exceed 0.003 µg/L.
E = Estimated (outside normal calibration limits)

Notes: The USGS Pesticide National Synthesis considered each well a distinct site in this analysis. Each well was represented by one sample: normally the first one taken, but possibly a later sample if the first sample was not analyzed for the full range of analytes.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Kolpin and Martin, 2003

The DCPA mono-acid degradate was not detected in ground water samples in undeveloped areas, mixed land use (major aquifer) settings, or urban areas (Exhibit 4-6). It was detected in 0.08% of ground water samples in agricultural settings. The 95th percentile concentrations were non-detects in all land use settings. The maximum ground water concentration in agricultural settings was 1.1 µg/L (Kolpin and Martin, 2003).

Exhibit 4-6: USGS National Synthesis Summary of NAWQA Monitoring of DCPA's Mono-Acid Degradate in Ambient Ground Water, 1992-2001

Land Use Type	No. of Wells	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,217	0.08%	<RL	<RL	1.1 µg/L
Mixed (Major Aquifer)	1,474	0.00%	<RL	<RL	<RL
Undeveloped	46	0.00%	<RL	<RL	<RL
Urban	619	0.00%	<RL	<RL	<RL

Abbreviations:
RL = Reporting limit. Reporting limits for dacthal mono-acid varied, but did not exceed 0.07 µg/L.

Notes: The USGS Pesticide National Synthesis considered each well a distinct site in this analysis. Each well was represented by one sample: normally the first one taken, but possibly a later sample if the first sample was not analyzed for the full range of analytes.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Kolpin and Martin, 2003

Bed Sediments and Biotic Tissue

The NAWQA program also investigated the occurrence of select organochlorine compounds, including DCPA, in bed sediments and biotic tissue. Sampling was conducted at 1,310 sites from 1992 to 2001. Method detection limits were 5 µg/kg dry weight in sediment, and 5 µg/kg wet weight in tissue. Details regarding sampling techniques and analytical methods are described by Nowell (2003). Organochlorines can be present in biotic tissue and in bed sediments of aquatic systems even when they are undetectable in the water column using conventional methods. The occurrence of a toxic compound in stream sediments is pertinent to drinking water concerns because some desorption of the compound from sediments into water, albeit at low rates, may be expected to occur through equilibrium reactions.

Exhibit 4-7 presents the NAWQA occurrence data for DCPA in bed sediment. These data indicate that DCPA occurred in bed sediment at detection frequencies ranging from 0.0% in urban settings to 0.5% in undeveloped settings, 0.6% in mixed land use settings, and 1.8% in agricultural land use settings. The 95th percentile concentrations in all land use settings were non-detects. The highest concentration, 33.7 µg/kg dry weight, was found in a mixed land use setting (Nowell, 2003).

Exhibit 4-7: USGS National Synthesis Summary of NAWQA Monitoring of DCPA in Bed Sediment, 1992-2001

Land Use Type	No. of Sites	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	282	1.8%	<RL	<RL	25 µg/kg
Mixed	338	0.6%	<RL	<RL	33.7 µg/kg
Undeveloped	224	0.5%	<RL	<RL	5 µg/kg
Urban	166	0.0%	<RL	<RL	<RL

Abbreviations:
RL = Reporting limit. Reporting limits for DCPA varied, but did not exceed 5 µg/kg.

Notes: For bed sediment, all weights are dry weights.

Most sites were sampled only once. In the case of sites sampled multiple times, USGS used a single sample (the earliest sample with complete data for key analytes) to represent each site in this analysis.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Nowell, 2003

In whole fish, DCPA detection frequencies ranged from 1.9% in undeveloped settings to 2.0% in urban settings, 4.5% in mixed settings, and 5.0% in agricultural settings (Exhibit 4-8). The 95th percentile concentrations in all settings were non-detects. The highest concentration, 78 µg/kg wet weight, was found in an agricultural setting (Nowell, 2003).

Exhibit 4-8: USGS National Synthesis Summary of NAWQA Monitoring of DCPA in Whole Fish, 1992-2001

Land Use Type	No. of Sites	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	204	5.0%	<RL	<RL	78 µg/kg
Mixed	207	4.5%	<RL	<RL	63 µg/kg
Undeveloped	162	1.9%	<RL	<RL	32 µg/kg
Urban	100	2.0%	<RL	<RL	8.5 µg/kg

Abbreviations:
RL = Reporting limit. Reporting limits for DCPA varied, but did not exceed 5 µg/kg.

Notes:
For whole fish, all weights are wet weights.

Most sites were sampled only once. In the case of sites sampled multiple times, USGS used a single sample (from the first year of sampling, the earliest sample of the variety of fish most often sampled in that Study Unit) to represent each site in this analysis.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Nowell, 2003

EPA Summary Analysis of NAWQA Data

Whereas the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination, including DCPA and the mono-acid degradate. Detection frequencies were simply computed as the percentage of samples and sites with detections (i.e., with at least one result equal to or greater than the reporting limit). Note that reporting limits were not uniform. Sample detections can be biased by frequent sampling in areas with high (or low) occurrence. Calculating the percentage of sites with detections can reduce this bias. For more details on the data set and the EPA analysis, see Chapter 2.

The results of the EPA analysis are presented in the following two Exhibits. Overall, DCPA was detected in 10.9% of samples and at 6.3% of sites. DCPA was detected more frequently in surface water than in ground water, and the highest concentration (100 µg/L) was found in surface water.

Exhibit 4-9: EPA Summary Analysis of DCPA Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	14,872	15.1%	1,907	21.0%	0.000004	0.003	0.078	0.99	100
ground water	6,080	0.8%	5,211	0.8%	0.0006	0.002	0.011	10	10
all sites	20,952	10.9%	7,118	6.3%	0.000004	0.003	0.0756	1	100

[1] RLs (Reporting Limits) for DCPA varied but did not exceed 0.003 µg/L. See Chapter 2 for more information.

Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible.

The DCPA mono-acid degradate was detected in 0.2% of samples and at 0.3% of sites. The DCPA mono-acid degradate was detected more frequently in surface water than in ground water, and the highest concentration (1.2 µg/L) was found in surface water.

Exhibit 4-10: EPA Summary Analysis of DCPA Mono-Acid Degradate Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	5,535	0.3%	894	1.1%	0.007	0.11	1.2	1.2	1.2
ground water	4,019	0.05%	3,645	0.1%	0.0395	0.5698	1.1	1.1	1.1
all sites	9,554	0.2%	4,539	0.3%	0.007	0.11	1.2	1.2	1.2

[1] RLs (Reporting Limits) for DCPA mono-acid varied but did not exceed 0.07 µg/L. See Chapter 2 for more information.

Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible.

Pesticide Reregistration Studies

Several small-scale studies on DCPA occurrence began in 1992 in support of DCPA reregistration. The studies are summarized in EPA's Reregistration Eligibility Decision for DCPA (USEPA, 1998).

For ground water, two monitoring studies were conducted. In New York State, a total of 29.4 pounds a.i. per acre were applied in three applications to a turf crop. Nine wells were tested over 17 months for detection of DCPA and its two metabolites. In California, a total of 18.2 pounds a.i. per acre were applied in two applications to an onion crop. Eight wells were tested over 22 months for detection of DCPA residues. In both ground water studies, the detection limit was 0.1 µg/L (USEPA, 1998).

Detected concentrations of DCPA and its two degradates were summed together. The New York site had an average sum of 50.36 µg/L for nine wells, and the California site had an average sum of 12.75 µg/L for eight wells (USEPA, 1998).

4.3.3 Drinking Water Occurrence

Nationally representative data on the occurrence of DCPA mono-acid and di-acid degradates in drinking water have been collected by large and small public water systems in accordance with EPA's first Unregulated Contaminant Monitoring Regulation (UCMR 1). For details on the UCMR 1, see Chapter 2 and USEPA (2006). Supplemental data from State and regional studies provide an indication of DCPA occurrence, and the occurrence of its most common degradates, in high-use areas.

UCMR 1

UCMR 1 monitoring was conducted primarily between 2001 and 2003, though some results were not collected and reported until as late as 2005. As List 1 contaminants, DCPA mono- and di-acid degradates were scheduled to be monitored by all large community water systems (CWSs) and non-transient non-community water systems (NTNCWSs) and a statistically representative sample of small CWSs and NTNCWSs. The data presented in this report reflect UCMR 1 analytical samples submitted and quality-checked under the regulation as of July 2005. DCPA degradate data were collected and submitted by 797 (99.6 percent) of the 800 small systems selected for the small system sample and 3,071 (99.1 percent) of the 3,100 large systems defined as eligible for the UCMR 1 large system census.

Because the analytical method approved for UCMR 1 use does not distinguish between the two degradates, they are measured and reported in aggregate. The DCPA degradate data have been analyzed at the level of simple detections (at or above the minimum reporting level, ≥ MRL, or ≥ 1 µg/L), exceedances of the HRL (> HRL, or > 70 µg/L), and exceedances of one-half the value of the HRL (> ½ HRL, or > 35 µg/L). Results of the analysis are presented in Exhibits 4-11 and 4-12.

Among small systems, DCPA degradate detections (≥ MRL, or ≥ 1 µg/L) were reported by 2.13% of public water systems (PWSs), representing 3.19% of the population served, equivalent to approximately 1.1 million people nationally. All but one of these systems was served by ground water. Only a single small system had a concentration greater than half the Health Reference Level (> ½ HRL, or > 35 µg/L) and the full HRL (> HRL, or > 70 µg/L); this ground water system represented 0.13% of small PWSs and 0.02% of the population served by them, equivalent to 113,000 persons nationally.

Among large systems, 158 systems (5.14%) had detections (≥ MRL, or ≥ 1 µg/L), affecting approximately 11.2 million people (5.05% of the population served). Most of these were ground water systems. A single large system had a concentration greater than half the Health Reference Level (> ½ HRL, or > 35 µg/L); this surface water system represented 0.03% of large PWSs and 0.33% of the population served by them (approximately 738,000 people). No large systems had detections at concentrations greater than the HRL (> HRL, or > 70 µg/L).

Exhibit 4-11: Summary UCMR 1 Occurrence Statistics for DCPA Mono- and Di-Acid Degradates in Small Systems (Based on Statistically Representative National Sample of Small Systems)

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,272		--
Percent of Samples with Detections	1.16%		--
99th Percentile Concentration (all samples)	1.3 µg/L		--
Health Reference Level (HRL)	70 µg/L		--
Minimum Reporting Level (MRL)	1 µg/L		--
Maximum Concentration of Detections	190 µg/L		--
99th Percentile Concentration of Detections	190 µg/L		--
Median Concentration of Detections	1.8 µg/L		--
Total Number of PWSs	797		60,414
Number of GW PWSs	590		56,072
Number of SW PWSs	207		4,342
Total Population	2,760,570		45,414,590
Population of GW PWSs	1,939,815		36,224,336
Population of SW PWSs	820,755		9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs with Detections (≥ MRL)	17	2.13%	689
GW PWSs with Detections	16	2.71%	652
SW PWSs with Detections	1	0.48%	37
PWSs > 1/2 HRL	1	0.13%	373
GW PWSs > 1/2 HRL	1	0.17%	373
SW PWSs > 1/2 HRL	0	0.00%	0
PWSs > HRL	1	0.13%	373
GW PWSs > HRL	1	0.17%	373
SW PWSs > HRL	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	87,933	3.19%	1,118,000
Pop. Served by GW PWSs with Detections	86,433	4.46%	1,074,000
Pop. Served by SW PWSs with Detections	1,500	0.18%	44,000
Population Served by PWSs > 1/2 HRL	500	0.02%	113,000
Pop. Served by GW PWSs > 1/2 HRL	500	0.03%	113,000
Pop. Served by SW PWSs > 1/2 HRL	0	0.00%	0
Population Served by PWSs > HRL	500	0.02%	113,000
Pop. Served by GW PWSs > HRL	500	0.03%	113,000
Pop. Served by SW PWSs > HRL	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 4-12: Summary UCMR 1 Occurrence Statistics for DCPA Mono- and Di-Acid Degradates in Large Systems (Based on the Census of Large Systems)

Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	30,480	
Percent of Samples with Detections	2.41%	
99th Percentile Concentration (all samples)	2.3 µg/L	
Health Reference Level (HRL)	70 µg/L	
Minimum Reporting Level (MRL)	1 µg/L	
Maximum Concentration of Detections	39 µg/L	
99th Percentile Concentration of Detections	16 µg/L	
Median Concentration of Detections	2.0 µg/L	
Total Number of PWSs	3,071	
Number of GW PWSs	1,384	
Number of SW PWSs	1,687	
Total Population	222,054,801	
Population of GW PWSs	53,434,814	
Population of SW PWSs	168,619,987	
Occurrence by System	**Number**	**Percentage**
PWSs with Detections (≥ MRL)	158	5.14%
GW PWSs with Detections	107	7.73%
SW PWSs with Detections	51	3.02%
PWSs > 1/2 HRL	1	0.03%
GW PWSs > 1/2 HRL	0	0.00%
SW PWSs > 1/2 HRL	1	0.06%
PWSs > HRL	0	0.00%
GW PWSs > HRL	0	0.00%
SW PWSs > HRL	0	0.00%
Occurrence by Population Served		
Population Served by PWSs with Detections	11,220,836	5.05%
Pop. Served by GW PWSs with Detections	6,034,379	11.29%
Pop. Served by SW PWSs with Detections	5,186,457	3.08%
Population Served by PWSs > 1/2 HRL	738,337	0.33%
Pop. Served by GW PWSs > 1/2 HRL	0	0.00%
Pop. Served by SW PWSs > 1/2 HRL	738,337	0.44%
Population Served by PWSs > HRL	0	0.00%
Pop. Served by GW PWSs > HRL	0	0.00%
Pop. Served by SW PWSs > HRL	0	0.00%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

The following maps, based on UCMR 1 data, give an indication of the geographic distribution of DCPA degradate occurrence in drinking water. Exhibit 4-13 shows the distribution of States with at least one detection. Exhibit 4-14 shows the relative frequency of detection in those States. Exhibit 4-15 shows the distribution of States with HRL exceedances. Although detections of DCPA degradates were relatively widespread, only one State—Michigan—had a concentration in excess of the HRL.

Exhibit 4-13: Geographic Distribution of DCPA Degradates in UCMR 1 Monitoring – States With At Least One Detection At or Above the MRL (≥ 1 µg/L)

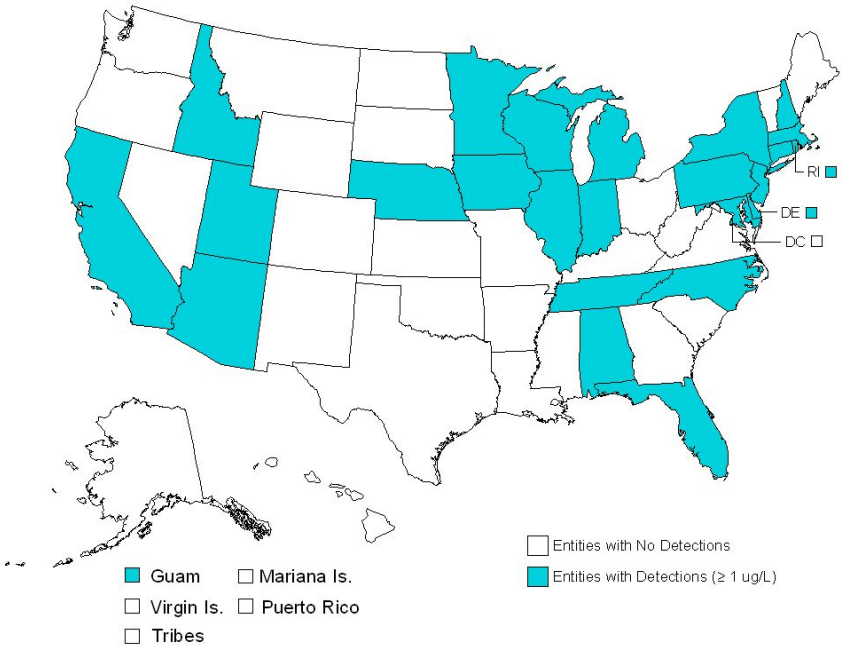
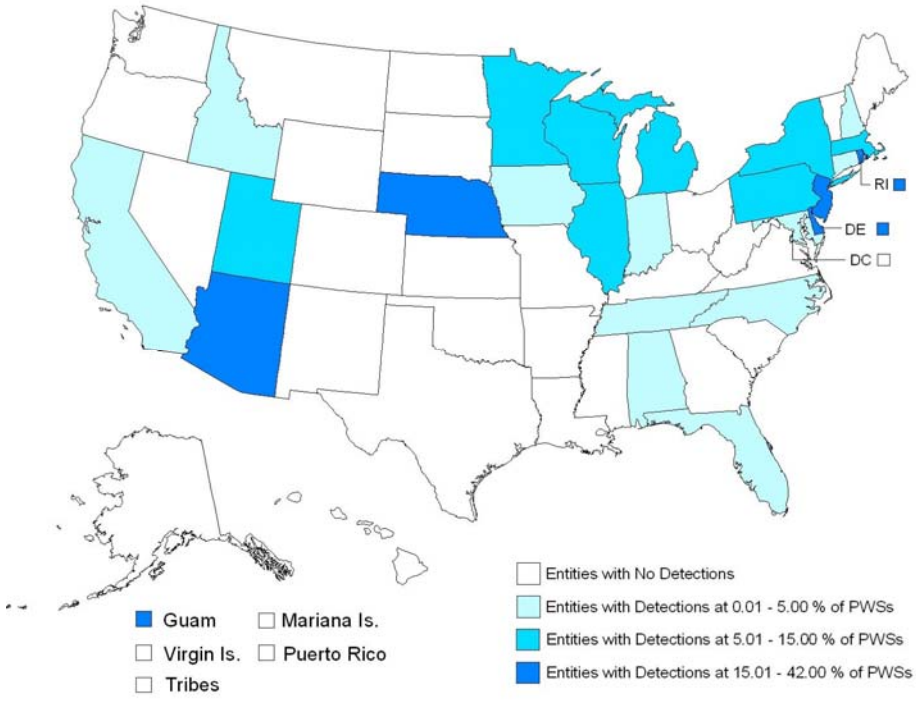


Exhibit 4-14: Geographic Distribution of DCPA Degradates in UCMR 1 Monitoring – Percentage of PWSs With At Least One Detection At or Above the MRL (≥ 1 µg/L), By State



Note: This map depicts UCMR 1 results from both small systems and large systems. The statistical selection of UCMR 1 small systems was designed to be representative at the national level, but not at the state level. Therefore, this map should only be considered a rough approximation of state-level patterns of contaminant occurrence.

Exhibit 4-15: Geographic Distribution of DCPA Degradates in UCMR 1 Monitoring – States With at Least One Detection Above the HRL (> 70 µg/L)

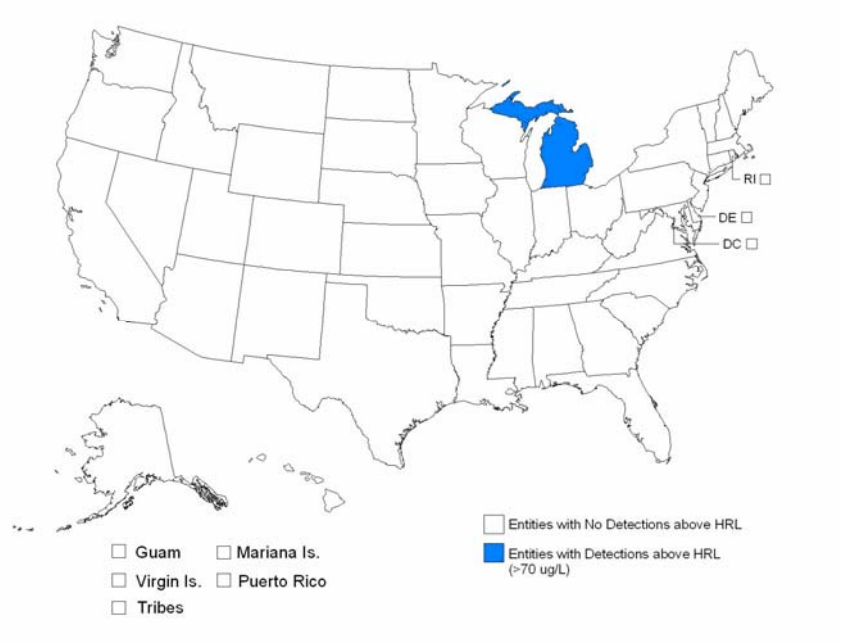
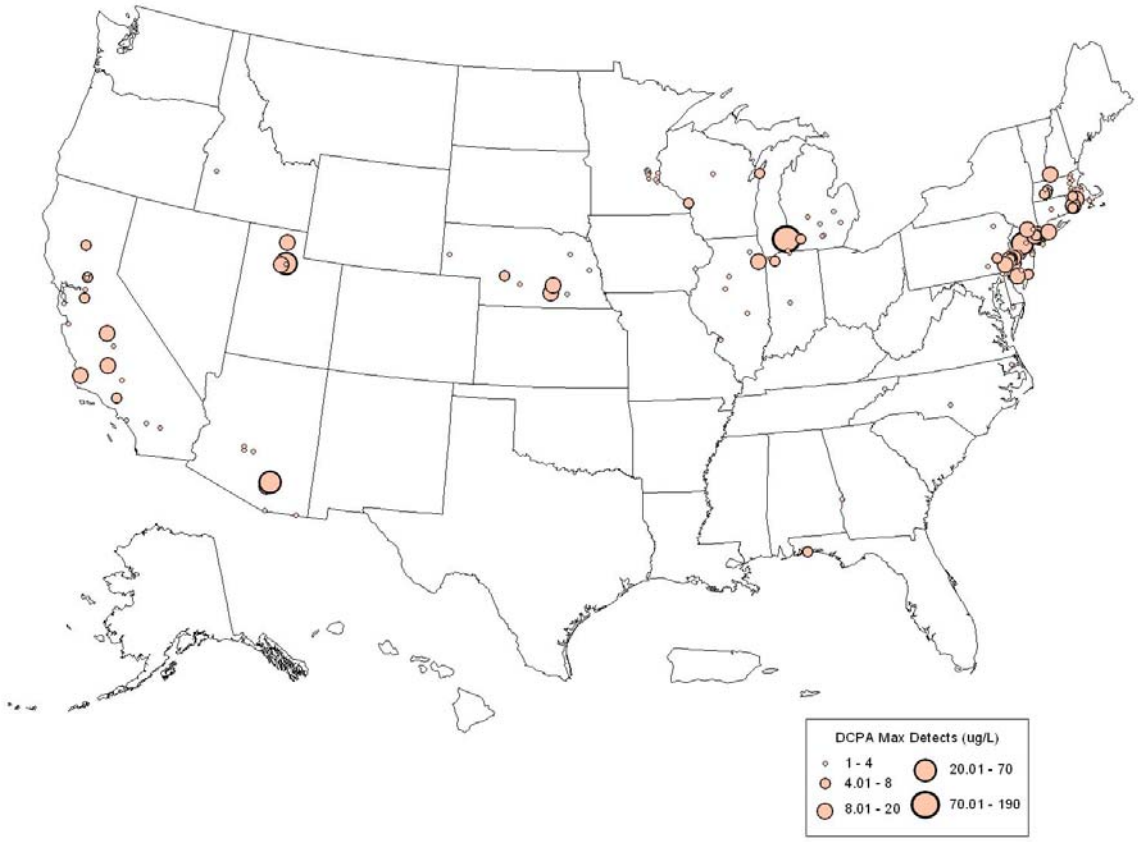


Exhibit 4-16 shows the location of PWSs with detections even more precisely. At this level of analysis, it is clear that the 172 detections of DCPA degradates are generally restricted to a few areas: California and Arizona, the Salt Lake City region, Nebraska, the Minneapolis-St. Paul metropolitan area, southern Lake Michigan, Philadelphia to New York City, and eastern Massachusetts and Rhode Island. The size of the dot represents the magnitude of the highest concentration detected at the system. The greatest grouping of high-concentration detections is in the Philadelphia to New York City vicinity. It is important to note, however, that all of the concentrations of DCPA degradates detected--with the exception of a single detection in Michigan--are below the HRL of 70 µg/L.

Exhibit 4-16: System-level Geographic Distribution of DCPA Degradates in UCMR 1 Monitoring – Maximum Concentration at Each System with Detections



For further analysis of UCMR 1 results for DCPA, see USEPA (2006).

Summary Analysis of Combined Large and Small System UCMR 1 Data

While the UCMR 1 data indicate that the DCPA degradates were the most commonly reported analytes in the monitoring survey (detected at an MRL of 1 µg/L in 772 samples from 175 of the 3,868 PWSs sampled), very few systems exceeded the health level of concern. PWSs with detections were found in 24 States and 1 Territory. The UCMR 1 data indicate that approximately 0.05 percent (or 2) of the 3,868 PWSs sampled had a detection of the DCPA degradates at levels greater than 35 µg/L, affecting approximately 0.33 percent of the population served (or 739,000 people from 225 million). Approximately 0.03 percent (or 1) of the 3,868 PWSs sampled have a detection of the DCPA degradates at levels greater than 70 µg/L, affecting less than 0.01 percent of the population served (or 500 people from 225 million). The average DCPA degradate concentration among detections was 3.48 µg/L and the median concentration was 2.00 µg/L.

Pesticides in Ground Water Database (PGWDB)

The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992). Most of the data are from drinking water wells. Since PGWDB data come from multiple sources, they should be interpreted with caution. Results might be biased high, because areas with suspected contamination are likely to have been sampled more frequently than pristine areas. For more information on PGWDB and the National Pesticide Survey (below), see Chapter 2.

According to the data compiled in the PGWDB, DCPA acid metabolites were detected in 59 of 118 wells (50.0 percent). Detections were found in all three states where the metabolites were investigated. Concentrations ranged from 0.223 to 0.308 µg/L in California, from 0.21 to 1.07 µg/L in Massachusetts, and from 1.0 to 431 µg/L in Oregon. The parent compound DCPA was detected in 5 of 2,033 wells sampled (0.25 percent). The parent compound was found in 3 out of 11 States where it was investigated. Concentrations ranged from 0.70 to 300 µg/L in California, and from 0.010 to 0.030 µg/L in Iowa; one Georgia well had a concentration of 99.0 µg/L. The HRL of 70 µg/L was exceeded in one State by degradates (Oregon) and in two States by the parent compound (California and Georgia) (USEPA, 1992).

In addition, the State of Washington sampled 81 wells for "DCPA and/or acid metabolites." Seven (8.64 percent) of the wells had detections. Concentrations ranged from 0.2 to 1.08 µg/L (USEPA, 1992).

National Pesticide Survey (NPS)

EPA collected samples from approximately 1,300 CWS wells and rural drinking water wells between 1988 and 1990 for the National Pesticide Survey (NPS). The survey was designed to provide a statistically reliable estimate of pesticide occurrence in the nation's drinking water wells. For details about NPS, see Chapter 2.

Of 126 pesticides and pesticide degradates monitored in the NPS, DCPA acid metabolites were the most commonly detected analyte. The survey projected that approximately 6.4 percent of CWSs and 2.5 percent of rural domestic wells nationwide were contaminated with DCPA acid metabolites at the level of the study's minimum reporting limit (0.10 µg/L) (USEPA, 1990b, 1990c). A correlation was found between the rate of DCPA application by urban applicators and on golf courses and detections of DCPA acid metabolites (USEPA, 1991a).

With a minimum reporting limit of 0.060 µg/L, the parent compound was not detected in the survey (USEPA, 1990d).

4.4 Technology Assessment

4.4.1 Analytical Methods

EPA evaluated the availability of analytical methods for all unregulated contaminants considered for UCMR 1 (64 FR 50556). Sources for these methods include publications by EPA

and by voluntary consensus standard organizations such as the American Society for Testing and Materials (ASTM), the Association of Official Analytical Chemists (AOAC), and the American Public Health Association (APHA).

Mono- and di-acid DCPA degradates are UCMR 1 List 1 contaminants which can be detected in drinking water by EPA Methods 515.1, 515.2, 515.3, and 515.4. The first two methods were approved for the monitoring of mono- and di-acid DCPA degradates in 1999 (64 FR 50556). The latter two were approved in a subsequent action (66 FR 2273). Methods 515.1, 515.2, and 515.4 do not distinguish between the two degradates, and give the total degradate concentration. Method 515.3 gives the total concentration of DCPA plus the two degradates. No EPA-approved method is currently available that distinguishes the mono-acid degradate from the di-acid degradate. The four EPA methods are generally similar, and involve hydrolyzation, extraction, derivatization, and cleanup steps before detection by gas chromatography with electron capture detection (GC/ECD). A full description of EPA Method 515.1 can be found in EPA's *Method for the Determination of Organic Compounds in Water* (USEPA, 1991b). Method 515.2 can be found in EPA's *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3* (USEPA, 1995a), and EPA Method 515.3 can be found in EPA's *Methods for the Determination of Organic and Inorganic Compounds in Drinking Water, Volume 1* (USEPA, 2000a). EPA Method 515.4 is available on EPA's Web site at: http://www.epa.gov/safewater/methods/met515_4.pdf (USEPA, 2000b). Additional methods approved for mono- and di-acid DCPA degradates include the ASTM Method D5317-93 (ASTM, 1996; 1998) and AOAC International 992.32 (AOAC, 1998).

The MDL and the average recovery for each analytical method that can be used for the analysis of DCPA degradates in water are included in the method descriptions below.[1]

EPA Method 515.1

In EPA Method 515.1 (Revision 4.0), *"Determination of Chlorinated Acids in Water by Gas Chromatography with an Electron Capture Detector*," a sample is adjusted to pH 12 with 6 N sodium hydroxide. The sample is then mixed for one hour to hydrolyze the derivatives. A solvent wash removes foreign organic material as well as the DCPA parent compound. Next, the sample is acidified, and the chlorinated acids are extracted with ethyl ether by shaking in a separatory funnel. Conversion of the extracted acids to methyl esters is achieved by utilizing diazomethane as the derivatizing agent. Extra derivatizing reagent is expelled, and the esters are separated and identified by GC/ECD (USEPA, 1991b).

[1] The Method Detection Limit (MDL) is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon the precision of a series of replicate measurements of the analyte at low concentrations. The MDL incorporates estimates of the accuracy of the determination. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

The average recovery is the fraction or percent concentration of a target analyte determined relative to the true or expected concentration from a sample containing a known amount of the target analyte. (This can result in apparent recovery values greater than 100 percent.)

The MDL for total mono- and di-acid DCPA degradates is 0.067 µg/L and the average recovery ranges from 74 to 81 percent depending on the method option used (USEPA, 1991b).

EPA Method 515.2

In EPA Method 515.2 (Revision 1.1), *"Determination of Chlorinated Acids in Water Using Liquid-Solid Extraction and Gas Chromatography with an Electron Capture Detector*," a sample is adjusted to pH 12 with 6 N sodium hydroxide. The sample is then mixed for one hour to hydrolyze the derivatives. A solvent wash removes foreign organic material as well as the DCPA parent compound. Next, the sample is acidified and the chlorinated acids are extracted with a 47 mm resin based extraction disk. The acids are eluted from the disk with 10 percent methanol in methyl tertiary butyl ether (MTBE). Conversion of the extracted acids to methyl esters is accomplished by utilizing diazomethane as the derivatizing agent. Extra derivatizing reagent is expelled, and the esters are separated and detected by GC/ECD (USEPA, 1995b).

The MDL demonstrated for the total mono- and di-acid degradates in Method 515.2 is 0.13 µg/L. The percent recovery ranges from 60 to 118 percent depending on the method option used (USEPA, 1995b).

EPA Method 515.3

In EPA Method 515.3 (Revision 1.0), *"Determination of Chlorinated Acids in Drinking Water By Liquid-Liquid Extraction, Derivatization and Gas Chromatography with Electron Capture Detection,"* a sample is adjusted to pH 12 with 4 N sodium hydroxide to hydrolyze the derivatives. Next, the aqueous sample is acidified and extracted with MTBE. The chlorinated acids, which have been separated into the organic phase, are then transformed to their methyl esters by one of two derivatization techniques. The first technique uses diazomethane as the methylating agent; the latter is a base-promoted esterification procedure and entails the addition of tetramethylammonium hydroxide and subsequent addition of methyl iodide. The target esters are then separated and identified by capillary column GC/ECD. Analytes are quantitated using procedural standard calibration (USEPA, 1996). Because there is no solvent wash step after the hydrolysis in this method, the parent compound DCPA will also be present in the sample extract, if it was present in the sample. Thus, this method does not distinguish between the mono- acid degradate, di-acid degradate, and parent compound, and the quantitative result from Method 515.3 represents the total of all of these forms. Data on the acid degradates collected for the UCMR 1 with Method 515.3 were required to be reanalyzed by Method 515.1, 515.2, or 515.4 if the initial result was greater than the MRL since Method 515.3 measures the total concentration of the parent and the two degradation products instead of only the degradation products.

The MDL for total DCPA and degradates using Method 515.3 is reported to range from 0.38 to 0.63 µg/L depending on the derivatization method used (USEPA, 1996). The recovery is reported to range from 88 to 123 percent depending on the derivatization method and the type of water used (USEPA, 1996).

EPA Method 515.4

In EPA Method 515.4, "*Determination of Chlorinated Acids in Drinking Water by Liquid-Liquid Microextraction, Derivatization, and Fast Gas Chromatography with Electron Capture Detection,"* a sample is adjusted to pH ≥12 with 4 N sodium hydroxide to hydrolyze the derivatives. Next, the sample is washed using a hexane and MTBE mixture for sample cleanup and to remove DCPA. Then the aqueous sample is acidified with sulfuric acid to a pH of less than 1 and extracted with MTBE. The chlorinated acids are then transformed to their methyl esters by derivatization with diazomethane. The target esters are then separated and identified by GC/ECD. Analytes are quantitated using the procedural standard calibration technique with an internal standard (USEPA, 2000b).

The primary column detection limit is 0.113 µg/L and the secondary column detection limit is 0.105 µg/L in reagent water (USEPA, 2000b). The average recovery ranges from 92 to 100 percent depending on the method option used (USEPA, 2000b).

4.4.2 Treatment Technologies

Treatment technology status does not influence the determination of whether or not a contaminant should be regulated. However, treatment technologies must be readily available before a contaminant can be regulated with a national primary drinking water regulation (NPDWR). There is no evidence that DCPA and its mono- and di-acid degradates are substantially removed by conventional treatments, such as coagulation/flocculation, sedimentation, and inert media filtration. Potential treatment technologies include membrane processes, activated carbon, and advanced oxidation.

Membranes are used in both low-pressure and high-pressure treatment processes. Low-pressure systems, which include microfiltration (MF) and ultrafiltration (UF), are most effective in removing particles and large molecules. High-pressure technologies, including nanofiltration (NF) and reverse osmosis (RO), are capable of removing dissolved organic contaminants. Both NF and RO are expected to be effective in removing DCPA and its degradates.

Granular activated carbon (GAC) treatment removes contaminants via the physical and chemical process of sorption: the contaminants attach to the carbon surface as water passes through the carbon bed. Activated carbon has a large sorption capacity for many water impurities, including synthetic organic chemicals, taste- and odor-causing compounds, and some species of mercury.

Adsorption capacity is typically represented by the Freundlich isotherm constant, with higher Freundlich (K) values indicating greater sorption potential. However, the Freundlich (K) values for DCPA and its mono- and di-acid degradates are not available. In general, contaminants containing halogen groups and contaminants with double bonds, as DCPA does, have a high affinity for carbon. In addition, compounds exhibiting low water solubility are expected to have high binding affinity for activated carbon. DCPA's degradates tend to be more soluble than the parent compound and therefore are expected to be less amenable to activated carbon treatment.

Advanced oxidation processes (AOPs) produce free hydroxyl radicals that have a high potential for oxidizing organic or inorganic contaminants in water. AOPs often employ combinations of oxidants, such as ultraviolet (UV) light, hydrogen peroxide, and ozone, for treatment that is more effective than one oxidant alone. AOPs are capable of treating many contaminants, including synthetic organic chemicals, taste- and odor-causing compounds, and inorganic contaminants such as sulfide, iron, and manganese (Najm and Trussell, 1999).

The susceptibility of a pesticide to oxidation may be inferred from aerobic soil metabolism data. Compounds with short aerobic metabolism half-lives are expected to be more amenable to treatment using AOPs. USEPA (1998) reports that the half-life of DCPA is between 18 to 37 days, while the half-life of the mono-acid degradate is shorter (2.8 days) and that of the di-acid degradate is longer (virtually no degradation in 300 days). These findings suggest that AOPs may be effective for the mono-acid degradate, less effective for the parent compound, and not effective for the di-acid degradate.

4.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate the DCPA mono-acid degradate and/or the DCPA di-acid degradate with a national primary drinking water regulation (NPDWR). Because these degradates appear to occur infrequently at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. While the Agency recognizes that these degradates have been detected in the PWSs monitored under the UCMR 1, only 1 PWS had a detect above the HRL.

The Agency encourages those States with public water systems that have detects for these degradates to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory for the DCPA parent to include the mono- and di-acid degradates, as well as any recent health information related to these compounds. The updated Health Advisory will provide information to any States with public water systems that may have DCPA degradates at levels above the HRL.

The Agency's preliminary regulatory determination for these contaminants is presented formally in the *Federal Register*.

4.6 References

American Society for Testing and Materials (ASTM). 1996. *Annual Book of ASTM Standards.* Volume 11.02. West Conshohocken, PA: American Society for Testing and Materials.

ASTM. 1998. *Annual Book of ASTM Standards.* Volume 11.02. West Conshohocken, PA: American Society for Testing and Materials.

Association of Official Analytical Chemist (AOAC) International. 1998. *Official Methods of Analysis of AOAC International.* 16th ed. 4th Revision. Volume I. Baltimore, MD: AOAC International.

Budavari, S. (ed.) 1989. *The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals*. Rahway, NJ: Merck and Co., Inc. (As cited in HSDB, 2004).

Doran. T. 1990. Response to EPA Review: Aerobic Soil Metabolism of Dacthal: Lab Project Number: 000/3EF/76/2083/001. (Unpublished study, as cited in USEPA, 1998).

Extension Toxicology Network (Extoxnet). 1996. Extension Toxicology Network Pesticide Information Profiles. Available on the Internet at: http://ace.ace.orst.edu/info/extoxnet/pips/DCPA.htm. Accessed August 14, 2001.

Glotfelty, D.E., A.W. Taylor, B.C. Turner, and W.H. Zoller. 1984. Volatilization of surface-applied pesticides from fallow soil. *Journal of Agricultural and Food Chemistry*. 32:638-643. (As cited in HSDB, 2004).

Hansch, C., A. Leo, and D. Hoekman. 1995. *Exploring QSAR Fundamentals and Applications in Chemistry and Biology*. Washington, DC: American Chemical Society. (As cited in HSDB, 2004).

Hazardous Substances Data Bank (HSDB). 2004. Search for Dimethyl Tetrachloroterephthalate. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed November 1, 2004.

ISK Biotech Corporation. 1993. MRID No. 42731001, 42998401 HED No. 010513. (Unpublished study, as cited in USEPA, 1994).

Klopman, G., D. Fercu, and H.S. Rosenkranz. 1996. The carcinogenic potential of dacthal and its metabolites. *Environmental Toxicology and Chemistry*. Vol. 15, No. 2. pp. 80-84.

Kolpin, D.W. and J.D. Martin. 2003. "Pesticides in Ground Water: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestgw/Pest-GW_2001_Text.html. Accessed August 24, 2004.

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt. 1990. *Handbook of Chemical Property Estimation Methods*. Washington, DC: American Chemical Society. (As cited in HSDB, 2004).

Martin, J.D., C.G. Crawford, and S.J. Larson. 2003. "Pesticides in Streams: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestsw/Pest-SW_2001_Text.html. Accessed August 24, 2004.

Najm, I. and R.R. Trussell. 1999. New and Emerging Drinking Water Treatment Technologies. *Identifying Future Drinking Water Contaminants*. Washington, DC: National Academy Press. pp. 220-243. Available to read on the Internet at: http://books.nap.edu/catalog/9595.html.

Nash, R.G. and T.J. Gish. 1989. Halogenated pesticide volatilization and dissipation from soil under controlled conditions. *Chemosphere*. 18:2353-2362. (As cited in USEPA, 1998).

National Center for Food and Agricultural Policy (NCFAP). 2003. National Pesticide Use Database. Available on the Internet at: http://www.ncfap.org/database/default.htm.

Nowell, L. 2003. "Organochlorine Pesticides and PCBs in Bed Sediment and Aquatic Biota from United States Rivers and Streams: Summary Statistics; Preliminary Results of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/sedbiota/. Accessed August 25, 2004. Last updated April 10, 2003.

Szalkowski, M., D. Stallard, and J. Killeen. 1980. Accumulation, Distribution, and Depuration of Carbon 14|-Dacthal (DS-893) in Bluegill Sunfish (Lepomis Macrochirus) under Flow Through Aquatic Conditions. Document No. 141-3EI-79-0138-001. Unpublished study prepared by Analytical Bio Chemistry Laboratories, Inc. pp. 40. (As cited in USEPA, 1998).

Szalkowski, M., J. Marciniszyn, and J. Killeen. 1981. Characterization and Quantitation of (Carbon-14)-Residues in Water and Fish Exposed to (Carbon-14)-Dimethyl 2,3,5,6-Tetrachloroterephthalate (Dacthal, DS-893) in a Flow Through Aquatic System. Document No. 142-4EI-81-0017-002. Unpublished study prepared by Analytical Bio Chemistry Laboratories, Inc. pp. 80. (As cited in USEPA, 1998).

Thelin, G.P. and L.P. Gianessi. 2000. *Method for Estimating Pesticide Use for County Areas of the Conterminous United States.* U.S. Geological Survey Open-File Report 00-250, 62 p. Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/ofr00250/ofr00250.pdf.

Tomlin, C.D.S. (ed.). 1997. *The Pesticide Manual – World Compendium*. 11th edition. Surrey, England: British Crop Protection Council. (As cited in HSDB, 2004).

USEPA. 1990a. *National Survey of Pesticides in Drinking Water Wells. Phase I Report.* EPA Report 570-9-90-015.

USEPA. 1990b. *National Pesticide Survey: DCPA Acid Metabolites*. EPA Report 570-9-90-NPS9. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS9.]

USEPA. 1990c. *National Pesticide Survey: Summary Results of EPA's National Survey of Pesticides in Drinking Water Wells*. EPA Report 570-9-90-NPS5. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS5.]

USEPA. 1990d. *National Pesticide Survey: Survey Analytes.* EPA Report 570-9-90-NPS2. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS2.]

USEPA. 1991a. *National Pesticide Survey: Update and Summary of Phase II Results*. EPA Report 570-9-91-021. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570991021.]

USEPA. 1991b. Method 515.1 - Determination of Chlorinated Acids in Water by Gas Chromatography with an Electron Capture Detector. Revision 4.0. In: *Methods for the Determination of Organic Compounds in Drinking Water*. EPA Report 600-4-88-039. December 1988, Revised July 1991.

USEPA. 1992. *Pesticides in Ground Water Database: A Compilation of Monitoring Studies, 1971-1991. National Summary.* EPA Report 734-12-92-001. Available on the Internet at: http://nepis.epa.gov/pubtitleOPPTS.htm. [Search for document number 7341292001.]

USEPA. 1994. Integrated Risk Information System (IRIS), Dacthal. August 1994. Available on the Internet at: http://www.epa.gov/iris/subst/0221.htm. Accessed February 2, 2005.

USEPA. 1995a. *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1995b. Method 515.2 - Determination of Chlorinates Acids in Water Using Liquid-Solid Extraction and Gas Chromatography with an Electron Capture Detector. Revision 1.1. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1996. Method 515.3 - Determination of Chlorinated Acids in Drinking Water By Liquid-Liquid Extraction, Derivatization and Gas Chromatography with Electron Capture Detection. Revision 1.0. In: *Methods for the Determination of Organic and Inorganic Compounds in Drinking Water, Volume 1.* EPA Report 815-R-00-014.

USEPA. 1997. *Pesticides Industry Sales And Usage: 1994 and 1995 Market Estimates*. EPA Report 733-R-97-002. Available on the Internet at: http://www.epa.gov/oppbead1/pestsales/95pestsales/market_estimates1995.pdf.

USEPA. 1998. Reregistration Eligibility Decision (RED)--DCPA. EPA Report 738-R-98-005. Washington, DC: Office of Prevention, Pesticides and Toxic Substances. November 1998. Available on the Internet at: http://www.epa.gov/oppsrrd1/REDs/0270red.pdf.

USEPA. 1999. *Pesticides Industry Sales And Usage: 1996 and 1997 Market Estimates*. EPA Report 733-R-99-001. Available on the Internet at: http://www.epa.gov/oppbead1/pestsales/97pestsales/market_estimates1997.pdf.

USEPA. 2000a. *Methods for the Determination of Organic and Inorganic Compounds in Drinking Water, Volume 1*. EPA 815-R-00-014. National Technical Information Service. NTIS PB2000-106981. Springfield, VA: U.S. Department of Commerce.

USEPA. 2000b. Method 515.4 - *Determination of Chlorinated Acids in Drinking Water By Liquid-Liquid Microextraction, Derivatization, and Fast Gas Chromatography with Electron*

Capture Detection. Revision 1.0. Available on the Internet at: http://www.epa.gov/safewater/methods/met515_4.pdf.

USEPA. 2002. *Pesticides Industry Sales And Usage: 1998 and 1999 Market Estimates*. EPA Report 733-R-02-001. Available on the Internet at: http://www.epa.gov/oppbead1/pestsales/99pestsales/market_estimates1999.pdf.

USEPA. 2004. *Memorandum - TPA (tetrachloroterephthalic acid)-metabolite of DCPA (Dacthal). Evaluation of Potential for Carcinogenicity*. Office of Prevention, Pesticide, and Toxic Substances. May 25, 2004.

USEPA. 2006. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

United States Geological Survey (USGS). 2004. 1997 Pesticide Use Maps. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pesticide_use_maps_1997/, accessed August 17, 2004, last updated August 14, 2003.

Wauchope, R.D., T.M. Buttler, A.G. Hornsby, P.W.M. Augustijn Beckers, and J.P. Burt. 1992. SCS/ARS/CES pesticide properties database for environmental decision-making. *Review of Environmental Contamination and Toxicology*. 123:1-155. (As cited in Extoxnet, 1996).

Yalkowsky, S.H. and R.M. Dannenfelser. 1992. AQUASOL Database of Aqueous Solubility. Version 5. Tucson, AZ: College of Pharmacy, University of Arizona. (As cited in HSDB, 2004).

Chapter 5:
DDE

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

1,1-Dichloro-2,2-bis(*p*-chlorophenyl)ethylene (DDE) is a primary metabolite of 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT), a pesticide once used to protect crops and eliminate disease-carrying insects in the U.S., until it was banned in 1973. DDE itself has no commercial uses and is only found in the environment as a result of contamination and/or breakdown of DDT. DDT production in the United States declined from 82 million kg in 1962 to 2 million kg in 1971. In smaller quantities, DDT production for export continued as late as the 1980s.

While DDE tends to adsorb strongly to surface soil and is fairly insoluble in water, it may enter surface waters from runoff that contains soil particles contaminated with DDE. In both soil and water, DDE is subject to photodegradation, biodegradation, and volatilization.

Limited data on DDE, mostly from a National Cancer Institute (NCI) bioassay, suggest that the liver is the primary target organ in mammalian species. However, the NCI study did not evaluate a full array of noncancer endpoints. There is an RfD of 0.0005 mg/kg/day for the parent pesticide DDT based on a no-observed-adverse-effect level (NOAEL) of 0.05 mg/kg/day from a dietary subchronic study. In this study, liver lesions were identified at a lowest-observed-adverse-effects level (LOAEL) of 0.25 mg/kg/day. Data on DDT identify effects on the nervous and hormonal systems as adverse effects that might also be seen with DDE because it is one of DDT's primary metabolites. The limited data for DDE suggest that any effects on the nervous system are less severe than those seen with DDT.

Based on animal studies, DDE is classified as "likely to be carcinogenic to humans." This classification is based on increases in the incidence of liver tumors, including carcinomas, in two strains of mice and in hamsters after dietary exposure to DDE. For this regulatory determination, EPA calculated an oral slope factor of 1.67×10^{-1} $(mg/kg/day)^{-1}$, resulting in a one-in-a-million cancer-risk health reference level (HRL) of 0.2 µg/L.

There are some indications that DDE has an adverse impact on the immune system. Considerable evidence exists that DDE can act as an endocrine disruptor. Children and adolescents may be sensitive populations for exposure to DDE due to its endocrine disruption properties.

Data on the ambient occurrence of DDE are available from the first monitoring cycle (1992-2001) of the United States Geological Survey's (USGS's) National Ambient Water Quality Association (NAWQA) program. While the USGS detected DDE in both surface and ground waters, 95 percent of the samples from the various land use settings were less than 0.006 µg/L (the USGS reporting limit). The maximum surface water concentration, 0.062 µg/L (agricultural setting), and the maximum ground water concentration, 0.008 µg/L (agricultural setting), are both less than the HRL.

To evaluate the occurrence of DDE in the nation's drinking water, EPA included it as an analyte in the first Unregulated Contaminant Monitoring Regulation (UCMR 1). Because the HRL for DDE (0.2 µg/L) is lower than the minimum reporting level (MRL) used for monitoring

(0.8 μg/L), EPA used the MRL value to evaluate occurrence and exposure. The MRL is within the 10^{-4} to the 10^{-6} cancer risk range for DDE. In evaluating the UCMR 1 data, EPA found that only 1 of the 3,867 public water systems (PWSs) sampled (approximately 0.03 percent) had a detection of DDE, affecting approximately 0.01 percent of the population served.

EPA also consulted data on DDE monitoring in ambient and drinking water from other sources, including National Urban Runoff Program, the Pesticides in Ground Water Database, and the National Pesticide Survey.

The Agency has made a preliminary determination not to regulate DDE with a national primary drinking water regulation (NPDWR). Because DDE appears to occur infrequently at levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

EPA recognizes that DDE is listed as a probable human carcinogen. For this reason, the Agency encourages those States with public water systems that might have DDE above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

AOAC	Association of Official Analytical Chemists
APHA	American Public Health Association
ASTM	American Society for Testing and Materials
CAS	Chemical Abstracts Service
CCL 2	Second Contaminant Candidate List
CWS	Community Water System
DDD	1,1-dichloro-2,2-bis(p-chlorophenyl)ethane
DDE	1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene or p,p-dichlorodiphenyldichloroethylene
DDT	1,1,1-trichloro-2,2-bis(p-chlorophenyl) ethane
ECD	Electron Capture Detector
GAC	Granular Activated Carbon
GC	Gas Chromatography
GC/ECD	Gas Chromatography with Electron Capture Detection
GC/MS	Gas Chromatography with Mass Spectrometry
HRL	Health Reference Level
LOAEL	Lowest Observed Adverse Effect Level
LSE	Liquid-Solid Extraction
MDL	Method Detection Limit
MRL	Minimum Reporting Level
MTBE	Methyl tertiary-butyl ether
NAWQA	National Water Quality Assessment
NCI	National Cancer Institute
NOAEL	No Observed Adverse Effect Level
NPDWR	National Primary Drinking Water Regulation
NPL	National Priorities List
NPS	National Pesticide Survey
NTNCWS	Non-Transient Non-Community Water System
PGWDB	Pesticides in Ground Water Database
PWS	Public Water System
RfD	Reference Dose
RL	Reporting Limit
RO	Reverse Osmosis
UCMR 1	First Unregulated Contaminant Monitoring Regulation
USGS	United States Geological Survey

5 DDE

5.1 Definition

DDE (1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene), an organochlorine, is a primary metabolite of DDT (1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane) along with DDD (1,1-dichloro-2,2-bis(p-chlorophenyl)ethane). DDE, like DDT and related compounds, can exist in three isomeric forms based on the relative position of the chlorine substitution on the two chlorophenyl rings. The most prevalent isomer, 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene, is commonly known as *p,p'*-DDE. The name "DDE" usually refers to *p,p'*-DDE. *p,p'*-DDE is also given the following names: 4,4'-DDE; dichlorodiphenyl-dichloroethane, 1,1-dichloro-2,2-bis(p-chlorophenyl) ethylene, and 1,1'-(2,2-dichloroethylidene)bis(4-chlorobenzene) (ATSDR, 2002). A less common isomer, 1,1-dichloro-2-(o-chlorophenyl)-2-(p-chlorophenyl)ethylene, is known as *o,p'*-DDE or 2,4'-DDE.

The notation in this document follows the usage in each source: for instance, the National Water Quality Assessment (NAWQA) program uses "*p,p'*-DDE" where the first Unregulated Contaminant Monitoring Regulation (UCMR 1) uses "4,4'-DDE." The Chemical Abstracts Service (CAS) registry number for DDE is 72-55-9.

5.1.1 Properties and Sources

DDT is a pesticide that was once widely used to control insects on agricultural crops and insects that carried diseases such as malaria and typhus. All uses of DDT in the United States were banned on January 1, 1973 except for case-by-case emergency measures (Meister and Sine, 1999 as cited in ATSDR, 2002). However, DDT is still produced and used in other countries as an anti-malarial measure. In Mexico, production ended in 1997 and use was phased-out by 2000 under the North American Agreement on Environmental Cooperation (CEC, 2003). Unlike DDT, DDE has no commercial use and is only found in the environment as a result of contamination or breakdown of DDT. DDT that has entered the atmosphere via spraying or volatilization can travel long distances and contaminate soils and surface waters by both wet and dry deposition. In the soil, DDT biodegrades to DDE under unflooded (generally aerobic) conditions and to DDD under flooded (generally anaerobic) conditions (ATSDR, 2002). DDT is highly persistent in the environment with reported half-lives between 2 and 15 years (Extoxnet, 1994). Vapor-phase degradation of DDT as a result of reactions with hydroxyl radicals in the atmosphere can act much faster, with an estimated half life of 37 hours (ATSDR, 2002). Analytical studies suggest that degradation of the insecticide dicofol, and of impurities in dicofol, could be additional sources of DDE (Risebrough *et al*., 1986 as cited in ATSDR, 2002). Physical and chemical properties of DDE are summarized in Exhibit 5-1.

Exhibit 5-1: Physical and Chemical Properties of DDE

Identification	
CAS number	72-55-9
Molecular Formula	$C_{14}H_8Cl_4$
Physical and Chemical Properties	
Boiling Point	336 °C [1]
Melting Point	89 °C [1]
Molecular Weight	318.03 g/mol [1]
Log K_{oc}	4.70 [2]
Log K_{ow}	6.51 [1]
Water Solubility	0.12 mg/L at 25 °C [1]
Vapor Pressure	6.0×10^{-6} mm Hg at 25 °C [1]
Henry's Law Constant	2.1×10^{-5} atm-m^3/mol [1] 2.8×10^{-3} (dimensionless), predicted [3] 4.1×10^{-4} (dimensionless), from literature [3]
Freundlich Isotherm Constant (K)	18,000 (µg/g)(L/µg)$^{1/n}$ [4]

[1] *Howard and Meylan, 1997 (as cited in ATSDR, 2002)*

[2] *Sabljic, 1984 (as cited in ATSDR, 2002)*

[3] *Speth et al., 2001*

[4] *Dobbs and Cohen, 1980 (as cited in Speth et al., 2001)*

5.1.2 Environmental Fate and Behavior

DDE strongly adsorbs to soil particles, especially in moist soils. As a result of strong binding to soil, DDE tends to remain on the surface layer of soil with little leaching into the lower soil layers and ground water (ATSDR, 2002). DDE is known to photodegrade and biodegrade on soil surfaces or when adsorbed to sediment (Baker and Applegate, 1970; Lichtenstein and Schultz, 1959; Miller and Zepp, 1979, all as cited in ATSDR, 2002). However, only limited data are available on degradation rates. One study found that the soil half-life of DDE ranged from 151 to 271 days in thirteen countries, while in a fourteenth country, where the soil was extremely acidic, the half-life was greater than 672 days (ATSDR, 2002).

Because DDE is fairly insoluble in water (see Exhibit 5-1), it is transported in runoff water principally by adsorption to particulate matter (ATSDR, 2002). In water, DDE may photodegrade or biodegrade. When exposed to sunlight, DDE undergoes photoisomerization. Direct photolysis of DDE results in a water half-life of about 1 day in the summer and 6 days in

the winter (ATSDR, 2002). However, as a hydrophobic organochlorine, DDE can persist for long periods of time in aquatic sediments and in the tissue of aquatic biota (USGS, 2000).

Volatilization accounts for considerable loss of this compound from soil surfaces and water. In the atmosphere, DDE can exist in vapor phase and particulate phase. In the vapor phase, DDE reacts with photochemically-produced hydroxyl radicals, with an estimated half-life of 17 hours to 2 days (ATSDR, 2002; HSDB, 2003). Attached to particles, DDE can last much longer, and can be transported long distances and deposited by wet or dry deposition. Because of long-range global transport of DDT, DDE, and related compounds, primarily from warmer regions to colder regions (ATSDR, 2002; CEC, 2003), DDE contamination could still be of concern even in countries like the U.S. where DDT has not been used in decades.

5.2 Health Effects

DDE is not produced as a commercial product. This has limited the numbers of conventional studies that have been performed to assess toxicological properties. Limited data on DDE, mostly from a National Cancer Institute (NCI) bioassay, suggest that the liver is the primary target organ in mammalian species. However, the NCI study did not evaluate a full array of noncancer endpoints. There is a reference dose (RfD) of 0.0005 mg/kg/day for the parent pesticide DDT based on a "no-observed-adverse-effect level" (NOAEL) of 0.05 mg/kg/day from a dietary subchronic study (USEPA, 1996). In this study, liver lesions were identified at a "lowest-observed-adverse-effect level" (LOAEL) of 0.25 mg/kg/day. Data on DDT identify effects on the nervous and hormonal systems as adverse effects that might also be seen with DDE because it is one of DDT's primary metabolites. The limited data for DDE suggest that any effects on the nervous system are less severe than those seen with DDT. Endocrine effects from DDE are discussed below.

Based on animal studies DDE is likely to be carcinogenic to humans. This classification is based on increases in the incidence of liver tumors, including carcinomas, in two strains of mice and in hamsters after dietary exposure to DDE. Some epidemiological studies suggest a possible association of the levels of DDE in serum with breast cancer. However, other studies with similar methodologies do not show any association. DDE was mutagenic in mouse lymphoma L5178Y and Chinese hamster V79 cells but negative in the Ames assay. In the 1988 IRIS, EPA calculated an oral slope factor of 0.34 $(mg/kg/day)^{-1}$ for DDE (USEPA, 1988). For this regulatory determination, EPA calculated an oral slope factor from the same data set (from the 1988 IRIS) using the EPA 1999 Cancer Guidelines (USEPA, 1999). The revised slope factor is 1.67×10^{-1} $(mg/kg/day)^{-1}$ resulting in a one-in-a-million cancer-risk health reference level (HRL) of 0.2 µg/L.

There are some indications that DDE has an adverse impact on the immune system (Banerjee *et al.*, 1996 as cited in ATSDR, 2002). Oral exposures to 22 mg/kg/day for six weeks suppressed serum immunoglobin levels and antibody titers. Inhibition of leucocytes and macrophage migration were observed at the cellular level. Considerable evidence exists that DDE can act as an endocrine disruptor since it binds to the estrogen and androgen receptors. DDE has a stronger affinity for the androgen receptor than for the estrogen receptor. It competes with testicular hormones for the androgen receptor leading to receptor-related changes in gene expression (Kelce *et al.*, 1995 as cited in ATSDR, 2002).

EPA evaluated whether health information is available regarding the potential effects on children and other sensitive populations. Children and adolescents may be sensitive populations for exposure to DDE due to its endocrine disruption properties. Some data suggest that DDE can delay puberty in males (ATSDR, 2002).

5.3 Occurrence and Exposure

5.3.1 Use and Environmental Release

DDT is a pesticide that was once widely used to control insects on agricultural crops, and insects that carried diseases such as malaria and typhus. All uses of DDT in the United States were banned on January 1, 1973 except for case-by-case emergency measures (USEPA, 1972). DDT production in the United States declined from 82 million kg in 1962 to 2 million kg in 1971. In smaller quantities, DDT production for export continued as late as the 1980s (ATSDR, 2002; HSDB, 2003).

Unlike DDT, DDE has no commercial use. It is only found in the environment as a result of contamination or breakdown of DDT. DDT that has entered the atmosphere via spraying or volatilization can contaminate soils and surface waters by both wet and dry deposition. In soil, DDT biodegrades to DDE under unflooded (generally aerobic) conditions and to DDD (dichlorodiphenyldichloroethane) under flooded (generally anaerobic) conditions (ATSDR, 2002).

Among the 1,613 hazardous waste sites in the United States and its territories that have been considered as candidates for inclusion in EPA's National Priorities List (NPL), at least 441 are known to be contaminated with DDT, DDE, and/or DDD. *p,p'*-DDE was found at 219 of these sites. While not specifically targeted, *o,p'*-DDE was also present in at least four sites. Of the 441 hazardous waste sites in which DDT, DDE, or DDD was detected, the contaminants were identified in air samples at 32 sites, in surface water samples at 101 sites, in ground water samples at 247 sites, and in sediment samples at 305 sites (HazDat, 2002 as cited in ATSDR, 2002).

5.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are the source of most drinking water. Data on the occurrence of DDE in ambient surface and ground water, as well as in bed sediment and biotic tissue, are available from the NAWQA program of the United States Geological Survey (USGS). For details on this program, see the discussion of NAWQA in Chapter 2. NAWQA data have been analyzed independently by USGS and EPA. Supplementary data are available from EPA's Nationwide Urban Runoff Program.

NAWQA National Pesticide Synthesis

Surface Water and Ground Water

Under the NAWQA program, USGS monitored *p,p'*-DDE between 1992 and 2001 in representative watersheds and aquifers across the country. Reporting limits in surface water and ground water varied but did not exceed 0.006 µg/L.

In surface water (Exhibit 5-2), *p,p'*-DDE was detected at frequencies ranging from 1.68% of samples in urban settings to 3.66% in undeveloped settings, 4.84% in agricultural settings, and 6.14% in mixed land use settings. The 95th percentile concentrations were below the reporting limit in all land use settings. The highest detected concentration, 0.062 µg/L, occurred in an agricultural setting (Martin *et al.*, 2003).

Exhibit 5-2: USGS National Synthesis Summary of NAWQA Monitoring of *p,p'*-DDE in Ambient Surface Water, 1992-2001

Land Use Type	No. of Samples (and No. of Sites)	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,885 (78)	4.84%	<RL	<RL	0.062 µg/L
Mixed	1,021 (47)	6.14%	<RL	<RL	0.009 µg/L
Undeveloped	60 (4)	3.66%	<RL	<RL	0.002 µg/L
Urban	900 (33)	1.68%	<RL	<RL	0.007 µg/L

Abbreviations:
RL = Reporting limit. Reporting limits for p,p'-DDE varied, but did not exceed 0.006 µg/L.

The USGS Pesticide National Synthesis used one year of data, generally the year with the most sampling results, to represent each site in this analysis. The sampling results were time-weighted, to eliminate bias from more frequent sampling at certain times of year. Detection Frequencies and Percentile Concentrations can be interpreted as representing annual occurrence. For instance, the detection frequency can be thought of as the percent of the year in which detections are found at a typical site in this land use category, and the 95th percentile concentration can be though of as a concentration that is not exceeded for 95% of the year at a typical site in this land use category.

Source: Martin et al., 2003

In ground water (Exhibit 5-3), *p,p'*-DDE detection frequencies ranged from 2.65% of samples in mixed land use settings (major aquifers) to 3.26% in agricultural settings, 3.96% in urban settings, and 7.46% in undeveloped settings. The 95th percentile concentrations were below the reporting limit in all land use settings. The highest detected concentration, 0.008 µg/L, was found in an agricultural setting (Kolpin and Martin, 2003).

Exhibit 5-3: USGS National Synthesis Summary of NAWQA Monitoring of *p,p'*-DDE in Ambient Ground Water, 1992-2001

Land Use Type	No. of Wells	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,443	3.26%	<RL	<RL	0.008 µg/L
Mixed (Major Aquifer)	2,716	2.65%	<RL	<RL	0.006 µg/L
Undeveloped	67	7.46%	<RL	<RL	0.002 µg/L
Urban	834	3.96%	<RL	<RL	0.005 µg/L

Abbreviations:
RL = Reporting limit. Reporting limits for p,p'-DDE varied, but did not exceed 0.006 µg/L.

The USGS Pesticide National Synthesis considered each well a distinct site in this analysis. Each well was represented by one sample: normally the first one taken, but possibly a later sample if the first sample was not analyzed for the full range of analytes.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Kolpin and Martin, 2003

Bed Sediments and Biotic Tissue

The NAWQA program also investigated the occurrence of select organochlorine compounds, including both *p,p'*-DDE and *o,p'*-DDE, in bed sediments and biotic tissue. Sampling was conducted at 1,310 sites from 1992 to 2001. Method detection limits for both isomers were 1 µg/kg dry weight in sediment, and 5 µg/kg wet weight in tissue. Details regarding sampling techniques and analytical methods are described by Nowell (2003). Organochlorines can be present in biotic tissues and in bed sediments of aquatic systems even when they are undetectable in the water column using conventional methods. The occurrence of a toxic compound in stream sediments is pertinent to drinking water concerns because some desorption of the compound from sediments into water, albeit at low rates, may be expected to occur through equilibrium reactions.

In bed sediment (Exhibit 5-4), *p,p'*-DDE detection frequencies range from 22% of samples in undeveloped settings to 46% in mixed land use settings, 48% in agricultural settings, and 70% in urban settings. The 95th percentile concentrations in bed sediment were found to range from 3.5 µg/kg dry weight (undeveloped settings) to 28.9 µg/kg dry weight (agricultural settings). The highest concentration, 440 µg/kg dry weight, was found in a mixed land use setting (Nowell, 2003).

Exhibit 5-4: USGS National Synthesis Summary of NAWQA Monitoring of *p,p'*-DDE in Bed Sediment, 1992-2001

Land Use Type	No. of Sites	Detection Frequency in samples	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	282	48%	0.98 µg/kg	28.9 µg/kg	190 µg/kg
Mixed	338	46%	0.81 µg/kg	11.6 µg/kg	440 µg/kg
Undeveloped	224	22%	<RL	3.5 µg/kg	31 µg/kg
Urban	166	70%	2.15 µg/kg	23.9 µg/kg	111 µg/kg

Abbreviations:
RL = Reporting limit. Reporting limits for p,p'-DDE varied, but did not exceed 1 µg/kg.

For sediment, all weights are dry weights.

Most sites were sampled only once. In the case of sites sampled multiple times, USGS used a single sample (the earliest sample with complete data for key analytes) to represent each site in this analysis.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Nowell, 2003

NAWQA data indicate that the more common isomer, *p,p'*-DDE, occurs in fish tissue at detection frequencies ranging from 44% of samples in undeveloped settings to 89% in agricultural settings, 89% in urban settings, and 93% in mixed land use settings (Exhibit 5-5). The 95th percentile concentrations in fish tissue were found to range from 128 µg/kg wet weight (undeveloped settings) to 2,180 µg/kg wet weight (agricultural settings). The highest concentration, 7,300 µg/kg wet weight, was found in an agricultural setting (Nowell, 2003).

Exhibit 5-5: USGS National Synthesis Summary of NAWQA Monitoring of *p,p'*-DDE in Whole Fish, 1992-2001

Land Use Type	No. of Sites	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	205	89%	43.5 µg/kg	2,180 µg/kg	7,300 µg/kg
Mixed	206	93%	42 µg/kg	397 µg/kg	7,200 µg/kg
Undeveloped	162	44%	3.50 µg/kg	128 µg/kg	1,300 µg/kg
Urban	100	89%	36 µg/kg	190 µg/kg	450 µg/kg

Abbreviations:
RL = Reporting limit. Reporting limits for p,p'-DDE varied, but did not exceed 5 µg/kg.

For whole fish, all weights are wet weights.

Most sites were sampled only once. In the case of sites sampled multiple times, USGS used a single sample (from the first year of sampling, the earliest sample of the variety of fish most often sampled in that Study Unit) to represent each site in this analysis.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Nowell, 2003

In bed sediment (Exhibit 5-6), *o,p'*-DDE detection frequencies range from 0% of samples in undeveloped settings to 1.6% in mixed land use settings, 2.6% in agricultural settings, and 3.7% in urban settings. The 95th percentile concentrations in bed sediment were less than the reporting limit in all land use settings. The highest concentration, 26.7 µg/kg dry weight, was found in an urban setting (Nowell, 2003).

Exhibit 5-6: USGS National Synthesis Summary of NAWQA Monitoring of *o,p'*-DDE in Bed Sediment, 1992-2001

Land Use Type	No. of Sites	Detection Frequency in samples	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	278	2.6%	<RL	<RL	4.4 µg/kg
Mixed	327	1.6%	<RL	<RL	22 µg/kg
Undeveloped	221	0.0%	<RL	<RL	<RL
Urban	164	3.7%	<RL	<RL	26.7 µg/kg

Abbreviations:
RL = Reporting limit. Reporting limits for o,p'-DDE varied, but did not exceed 1 µg/kg.

For sediment, all weights are dry weights.

Most sites were sampled only once. In the case of sites sampled multiple times, USGS used a single sample (the earliest sample with complete data for key analytes) to represent each site in this analysis.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Nowell, 2003

NAWQA data indicate that the less common isomer, *o,p'*-DDE, occurs in fish tissue at detection frequencies ranging from 0.0% of samples in undeveloped settings to 3.2% in mixed land use settings, 6.4% in urban settings, and 7.0% in agricultural settings (Exhibit 5-7). The 95th percentile concentrations in fish tissue were found to range from undetectable (undeveloped and mixed land use settings) to 10 µg/kg wet weight (agricultural settings). The highest concentration, 130 µg/kg wet weight, was found in a mixed land use setting (Nowell, 2003).

Exhibit 5-7: USGS National Synthesis Summary of NAWQA Monitoring of *o,p'*-DDE in Whole Fish, 1992-2001

Land Use Type	No. of Sites	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	204	7.0%	<RL	10 µg/kg	85 µg/kg
Mixed	206	3.2%	<RL	<RL	130 µg/kg
Undeveloped	162	0.0%	<RL	<RL	<RL
Urban	99	6.4%	<RL	6.9 µg/kg	22 µg/kg

Abbreviations:
RL = Reporting limit. Reporting limits for o,p'-DDE varied, but did not exceed 5 µg/kg.

For whole fish, all weights are wet weights.

Most sites were sampled only once. In the case of sites sampled multiple times, USGS used a single sample (from the first year of sampling, the earliest sample of the variety of fish most often sampled in that Study Unit) to represent each site in this analysis.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Nowell, 2003

EPA Summary Analysis of NAWQA Data

Whereas the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination, including DDE. Detection frequencies were simply computed as the percentage of samples and sites with detections (i.e., with at least one result equal to or greater than the reporting limit). Note that reporting limits were not uniform. Sample detections can be biased by frequent sampling in areas with high (or low) occurrence. Calculating the percentage of sites with detections can reduce this bias. For more details on the data set and the EPA analysis, see Chapter 2.

The results of the EPA analysis are presented in Exhibit 5-8. Overall, DDE was detected in 5.0% of samples and at 6.4% of sites. DDE was detected more frequently and at higher concentrations (maximum of 0.062 µg/L) in surface water.

Exhibit 5-8: EPA Summary Analysis of DDE Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	14,880	5.6%	1,907	13.8%	0.0001	0.0024	0.0168	0.026	0.062
ground water	6,079	3.3%	5,210	3.7%	0.0001	0.0014	0.0032	0.0056	0.0076
all sites	20,959	5.0%	7,117	6.4%	0.0001	0.00205	0.015	0.025	0.062

[1] RLs (Reporting Limits) for DDE varied but did not exceed 0.006 µg/L. For more information, see Chapter 2. Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible.

Nationwide Urban Runoff Program

A total of 86 urban runoff samples from 15 U.S. cities, collected between 1979 and 1982 in connection with EPA's National Urban Runoff Program, were analyzed by Cole *et al.* (1984). Neither DDE nor DDD were detected in any sample. DDT was detected in 1 percent of samples, at a concentration of 0.1 µg/L. Detection limits were not reported. For background to the National Urban Runoff Program, see Chapter 2.

5.3.3 Drinking Water Occurrence

Nationally representative data on 4,4'-DDE occurrence in drinking water have been collected by large and small public water systems in accordance with EPA's first Unregulated Contaminant Monitoring Regulation (UCMR 1). For further details on the UCMR 1, see Chapter 2 and USEPA (2006).

UCMR 1

UCMR 1 monitoring was conducted primarily between 2001 and 2003, though some results were not collected and reported until as late as 2005. As a List 1 contaminant, 4,4'-DDE was scheduled to be monitored by all large community water systems (CWSs) and non-transient non-community water systems (NTNCWSs) and a statistically representative sample of small CWSs and NTNCWSs. The data presented in this report reflect UCMR 1 analytical samples submitted and quality-checked under the regulation as of July 2005. 4,4'-DDE data were collected and submitted by 797 (99.6 percent) of the 800 small systems selected for the small system sample and 3,070 (99.0 percent) of the 3,100 large systems defined as eligible for the UCMR 1 large system census. 4,4'-DDE data have been analyzed at the level of simple detections (at or above the minimum reporting level, ≥ MRL, or ≥ 0.8 µg/L). Since the HRL of 0.2 µg/L is less than the MRL, the data are not analyzed at the level of the HRL or half the HRL.

EPA set the MRL for UCMR 1 contaminants based on the capability of analytical methods, not anticipated health levels. For many UCMR 1 contaminants, including DDE, the MRL was determined by multiplying by 10 the least sensitive method's minimum detection limit, or, when available, multiplying by 5 the least sensitive method's estimated detection limit (USEPA, 2000). MRLs were set approximately an order of magnitude higher than detection limits to ensure consistency, accuracy, and reproducibility of results. The MRL for DDE is within the risk range of 10^{-6} to 10^{-4} used by EPA to evaluate carcinogens (see Section 2.1.1).

Results of the analysis are presented in Exhibits 5-9 and 5-10. No detections of 4,4'-DDE were found in any samples from small systems. DDE was detected at a single large system; this ground water system represented 0.03% of large public water systems (PWSs) and 0.01% of the population served by them (approximately 18,000 people). The concentration of the single detection was 3 µg/L.

Exhibit 5-9: Summary UCMR 1 Occurrence Statistics for 4,4'-DDE in Small Systems (Based on Statistically Representative National Sample of Small Systems)

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,251		--
Percent of Samples with Detections	0.00%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	0.2 µg/L		--
Minimum Reporting Level (MRL)	0.8 µg/L		--
Maximum Concentration of Detections	< MRL		--
99th Percentile Concentration of Detections	< MRL		--
Median Concentration of Detections	< MRL		--
Total Number of PWSs Number of GW PWSs Number of SW PWSs	797 590 207		60,414 56,072 4,342
Total Population Population of GW PWSs Population of SW PWSs	2,760,570 1,939,815 820,755		45,414,590 36,224,336 9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only

Exhibit 5-10: Summary UCMR 1 Occurrence Statistics for 4,4'-DDE in Large Systems (Based on the Census of Large Systems)

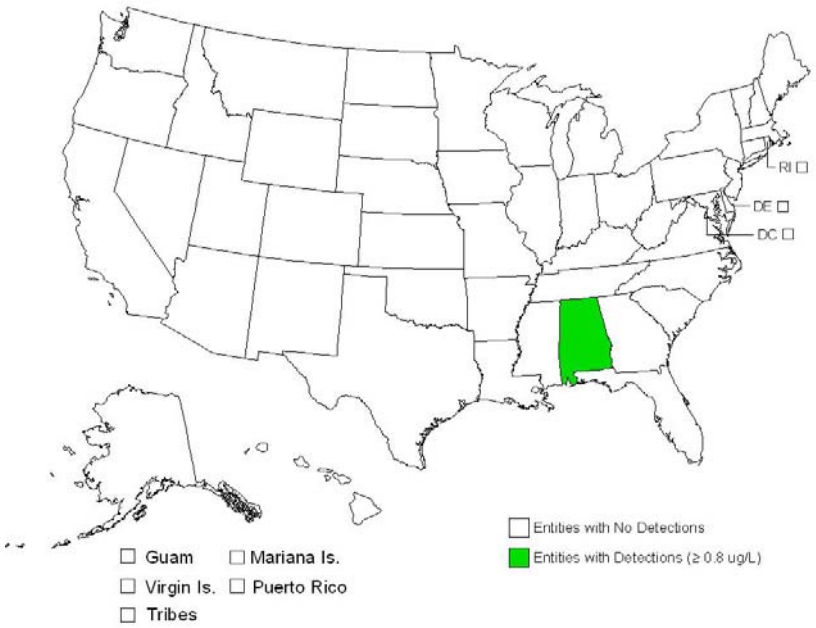
Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	30,383	
Percent of Samples with Detections	0.003%	
99th Percentile Concentration (all samples)	< MRL	
Health Reference Level (HRL)	0.2 µg/L	
Minimum Reporting Level (MRL)	0.8 µg/L	
Maximum Concentration of Detections	3 µg/L	
99th Percentile Concentration of Detections	3 µg/L	
Median Concentration of Detections	3 µg/L	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	3,070 1,376 1,694	
Total Population Population of GW PWSs Population of SW PWSs	223,371,547 53,313,206 170,058,341	
Occurrence by System	**Number**	**Percentage**
PWSs (GW & SW) with Detections (≥ MRL) GW PWSs with Detections SW PWSs with Detections	1 1 0	0.03% 0.07% 0.00%
Occurrence by Population Served		
Population Served by PWSs with Detections Pop. Served by GW PWSs with Detections Pop. Served by SW PWSs with Detections	17,670 17,670 0	0.01% 0.03% 0.00%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

DDE was only detected in one sample in all of the UCMR 1 sampling. This single detection was in a ground water sample taken in the State of Alabama (see Exhibit 5-11). Since only one system detected the contaminant, no further spatial analysis of this contaminant is presented.

Exhibit 5-11: Geographic Distribution of 4,4'-DDE in UCMR 1 Monitoring – States With at Least One Detection At or Above the MRL (≥ 0.8 µg/L)



Summary Analysis of Combined Large and Small System UCMR 1 Data

The UCMR 1 data indicate that approximately 0.03 percent (or 1) of the 3,867 PWSs sampled had a detection of DDE at the MRL of 0.8 µg/L, affecting approximately 0.01 percent of the population served (or 18,000 people from 226 million).

Pesticides in Ground Water Database (PGWDB)

The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992). Most of the data are from drinking water wells. Since PGWDB data come from multiple sources, they should be interpreted with caution. Results might be biased high, because areas with suspected contamination are likely to have been sampled more frequently than pristine areas. For further background on the PGWDB, see Chapter 2.

According to the data compiled in the PGWDB, DDE was detected in 34 of 2,918 wells (1.17 percent). The parent compound DDT was detected in 108 of 3,115 wells (3.47 percent), and the related compound DDD was detected in 35 of 2,647 wells (1.32 percent). DDE was found in 6 out of 17 States where monitoring was conducted. DDT was also found in 6 out of 17 States. DDD was found in 4 out of 17 States. DDE concentrations ranged from 0.010 to 0.090 µg/L in California, from 0.19 to 0.28 µg/L in Indiana, from 0.002 to 0.54 µg/L in Maine, and from 0.01 to 0.3 µg/L in South Carolina; one Connecticut well and one New Jersey well each had a concentration of 0.001 µg/L. The HRL of 0.2 µg/L was exceeded by DDE concentrations in three States: Indiana (maximum concentration of 0.28 µg/L), Maine (maximum concentration of 0.54 µg/L), and South Carolina (maximum concentration of 0.3 µg/L). The highest DDT and DDD concentrations were 3.3 µg/L and 1.040 µg/L, respectively (USEPA, 1992).

National Pesticide Survey (NPS)

EPA collected samples from approximately 1,300 CWS wells and rural drinking water wells between 1988 and 1990 for the National Pesticide Survey (NPS). The survey was designed to provide a statistically reliable estimate of pesticide occurrence in the nation's drinking water wells. For details about the NPS, see Chapter 2.

With a minimum reporting limit of 0.060 µg/L, DDE was not detected in the survey. DDT (with a reporting limit of 0.15 µg/L) and DDD (with a reporting limit of 0.13 µg/L) were also not detected (USEPA, 1990).

5.4 Technology Assessment

5.4.1 Analytical Methods

EPA evaluated the availability of analytical methods for all of the unregulated contaminants considered for UCMR 1, promulgated in 1999 (64 FR 50556). Sources for these methods include publications by EPA and by voluntary consensus standard organizations such as the American Society for Testing and Materials (ASTM), the Association of Official Analytical Chemists (AOAC), and the American Public Health Association (APHA).

DDE is a UCMR 1 List 1 contaminant that can be detected in drinking water by EPA Methods 508, 508.1 and 525.2. These methods were approved for the monitoring of DDE in 1999 (64 FR 50556). EPA Method 508 relies on liquid-liquid extraction followed by gas chromatography with electron capture detection (GC/ECD). EPA Method 508.1 relies on liquid-solid extraction (LSE) followed by GC/ECD. Like Method 508.1, Method 525.2 relies on LSE, but for detection it uses capillary column GC with mass spectrometry (GC/MS). Brief summaries of all three methods are provided below. Full descriptions can be found in EPA's *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3* (USEPA, 1995a). Additional methods approved for DDE include ASTM Method D5812-96 (ASTM, 1996; 1998) and AOAC 990.06 (AOAC, 1998).

The method detection limit (MDL) and the average recovery for each analytical method that can be used for the analysis of DDE in water are included in the method descriptions below.[1]

EPA Method 508

In EPA Method 508 (Revision 3.1), "*Determination of Chlorinated Pesticides in Water by Gas Chromatography with an Electron Capture Detector*," a measured volume of a water sample is solvent-extracted with methylene chloride by shaking in a separatory funnel or mechanical bumbling in a bottle. The methylene chloride extract is isolated, dried, and concentrated after solvent substitution with methyl tert-butyl ether (MTBE). The concentrated extract is then analyzed by capillary column GC/ ECD (USEPA 1995b).

The MDL for DDE in reagent water is 0.0025 µg/L and the average recovery is 127 percent (USEPA, 1995b).

EPA Method 508.1

In EPA Method 508.1 (Revision 2.0), *"Determination of Chlorinated Pesticides, Herbicides, and Organohalides by Liquid-Solid Extraction and Electron Capture Chromatography*," the analytes are extracted by LSE (i.e., passing a water sample through a preconditioned disk or cartridge containing a solid matrix coated with a chemically bonded C_{18} organic phase). The analytes are eluted from the LSE disk or cartridge with small amounts of ethyl acetate and methylene chloride. The analytes are then concentrated by evaporation of some of the solvent. The concentrated extract is analyzed by injecting micro-liter amounts of the eluate into a high resolution fused silica capillary column of a GC/ECD system (USEPA, 1995c).

The MDL for DDE is 0.003 µg/L in reagent water, while the average recovery ranges from 80 to 96.5 percent depending on the spike concentration used (USEPA, 1995c).

EPA Method 525.2

EPA Method 525.2 (Revision 2.0), *"Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry*," also uses the LSE method as described above. Compounds eluting from the GC column are characterized by comparing their measured mass spectra and retention times to reference mass spectra and retention times (USEPA, 1995d).

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[1] The Method Detection Limit (MDL) is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon the precision of a series of replicate measurements of the analyte at low concentration. The MDL incorporates estimates of the accuracy of the determination. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

The average recovery is the fraction or percent concentration of a target analyte determined relative to the true or expected concentration from a sample containing a known amount of the target analyte. (This can result in apparent recovery values greater than 100 percent.)

The MDL for DDE in reagent water ranges from 0.054 to 0.075 µg/L depending on the method option used. The recovery for DDE is reported as 104 percent (USEPA, 1995d).

5.4.2 Treatment Technologies

Treatment technology status does not influence the determination of whether or not a contaminant should be regulated. However, treatment technologies must be readily available before a contaminant can be regulated with a national primary drinking water regulation (NPDWR). There is no evidence that DDE is substantially removed by conventional treatments, such as coagulation/flocculation, sedimentation, and inert media filtration. Potential treatment technologies include activated carbon and reverse osmosis.

Granular activated carbon (GAC) treatment removes contaminants via the physical and chemical process of sorption, by which the contaminants attach to the carbon surface as water passes through the carbon bed. Activated carbon has a large sorption capacity for many water impurities, including synthetic organic chemicals, taste- and odor-causing compounds, and some species of mercury.

Adsorption capacity is typically represented by the Freundlich isotherm constant, with higher Freundlich (K) values indicating greater sorption potential. Activated carbon is considered to be cost-effective for removing a particular contaminant if the Freundlich (K) value of the contaminant is above 200 µg/g $(L/\mu g)^{1/n}$ (Speth *et al.*, 2001). Dobbs and Cohen (1980 as cited in Speth *et al.*, 2001) determined that the Freundlich (K) value for DDE is 18,000 µg/g $(L/\mu g)^{1/n}$, which suggests that GAC is a promising treatment option.

Reverse osmosis (RO) is similar to other membrane processes, such as ultrafiltration and nanofiltration, in that water passes through a semi-permeable membrane. However, in the case of RO, the membrane is non-porous. RO involves the use of applied hydraulic pressure to oppose the osmotic pressure across the membrane, forcing the water from the concentrated-solution side to the dilute-solution side. The water dissolves into the membrane, diffuses across, then dissolves out into the permeate. Most inorganic and many organic contaminants are rejected by the membrane and will be retained in the concentrate.

USEPA (2001) reports that the organochlorine class of pesticides can be removed with 99.9 to 100 percent efficiency using a cellulose acetate membrane and 100 percent efficiency using a thin-film composite membrane. These results indicate that RO is a promising option for removal of DDE in drinking water.

5.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate DDE with a national primary drinking water regulation (NPDWR). Because DDE appears to occur infrequently at levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. DDE was detected in only one of the PWSs monitored under the UCMR 1 at a level greater than the MRL (0.8 µg/L), a concentration that is within the 10^{-4} to the 10^{-6} cancer risk range. In addition, ambient water data from the USGS indicate that

the maximum concentrations detected in surface and ground water were less than the HRL of 0.2 µg/L.

EPA recognizes that DDE is listed as a probable human carcinogen. For this reason, the Agency encourages those States with public water systems that might have DDE above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

5.6 References

American Society for Testing and Materials (ASTM). 1996. *Annual Book of ASTM Standards.* Volume 11.02. West Conshohocken, PA: American Society for Testing and Materials.

ASTM. 1998. *Annual Book of ASTM Standards.* Volume 11.02. West Conshohocken, PA: American Society for Testing and Materials.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological Profile DDT, DDE, and DDD*. Available on the Internet at: http://www.atsdr.cdc.gov/toxprofiles/tp35.html.

Association of Official Analytical Chemist (AOAC) International. 1998. *Official Methods of Analysis of AOAC International.* 16th ed. 4th Revision. Volume I. Baltimore, MD: AOAC International.

Baker R.D. and H.G. Applegate. 1970. Effect of temperature and ultraviolet graduation on the persistence of methyl parathion and DDT in soils. *Agronomy Journal.* 62:509-512. (As cited in ATSDR, 2002).

Banerjee, B.D., A. Ray, and S.T. Pasha. 1996. A comparative evaluation of immunotoxicity of DDT and its metabolites in rats. *Indian Journal of Experimental Biology*. 34:517-522. (As cited in ATSDR, 2002).

Cole, R.H., R.E. Frederick, R.P. Healy, and R.G. Rolan. 1984. Preliminary findings of the priority pollutant monitoring project of the Nationwide Urban Runoff Program. *Journal of the Water Pollution Control Federation*. 56(7):898-908.

Commission for Environmental Cooperation of North America (CEC). 2003. DDT no longer used in North America. Fact Sheet, June, 2003. Montreal, Quebec. Available on the Internet at: http://www.cec.org/files/pdf/POLLUTANTS/DDT_en.pdf, accessed February 8, 2006.

Dobbs, R.A. and J.M. Cohen. 1980. *Carbon Adsorption Isotherms for Toxic Organics*. EPA 600/8-80-023. Cincinnati, OH: Municipal Environmental Research Laboratory, USEPA. (As cited in Speth *et al.*, 2001).

Extoxnet. 1994. "Extension Toxicology Network Pesticide Information Profiles– DDT(dichlorodiphenyltrichloroethane)." Available on the Internet at: http://pmep.cce.cornell.edu/profiles/extoxnet/carbaryl-dicrotophos/ddt-ext.html. Accessed December 16, 2004.

Hazardous Substance Data Bank (HSDB). 2003. Search for DDT. Available on the Internet through Toxnet, sponsored by the National Institute Health Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed August 26, 2004.

Hazardous Substance Release/Health Effects Database (HazDat). 2002. Agency for Toxic Substance and Disease Registry (ATSDR). Atlanta, GA. (As cited in ATSDR, 2002).

Howard P, and W. Meylan (eds.) 1997. *Handbook of physical properties of organic chemicals*. Boca Raton, FL: CRC Press, Lewis Publishers. (As cited in ATSDR, 2002).

Kelce, W.R., C.R. Stone, S.C. Laws, L.E. Gray, J.A. Kemppainen, and E.M. Wilson. 1995. Persistent DDT metabolite p,p'-DDE is a potent androgen receptor antagonist. *Nature*. Vol. 375. pp. 581-585. (As cited in ATSDR, 2002).

Kolpin, D.W. and J.D. Martin. 2003. Pesticides in Ground Water: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestgw/Pest-GW_2001_Text.html, accessed August 24, 2004. Link to document from: http://ca.water.usgs.gov/pnsp/.

Lichtenstein, E. and K. Schulz. 1959. Persistence of some chlorinated hydrocarbon insecticides as influenced by soil types, rate of application and temperature. *Journal of Economic Entomology*. 52(1):124-131. (As cited in ATSDR, 2002).

Martin, J.D., C.G. Crawford, and S.J. Larson. 2003. Pesticides in Streams: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestsw/Pest-SW_2001_Text.html, accessed August 24, 2004. Link to document from: http://ca.water.usgs.gov/pnsp/.

Meister, R. and C. Sine. 1999. Farm Chemicals Handbook. Willoughby, OH; Meister Publication Company, C123-C124. (As cited in ATSDR, 2002).

Miller, G. and R. Zepp. 1979. Photoreactivity of aquatic pollutants sorbed on suspended sediments. *Environmental Science and Technology.* 3(7):860-863. (As cited in ATSDR, 2002).

Nowell, L. 2003. "Organochlorine Pesticides and PCBs in Bed Sediment and Aquatic Biota from United States Rivers and Streams: Summary Statistics; Preliminary Results of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/sedbiota/. Accessed August 25, 2004. Last updated April 10, 2003.

Risebrough, R., W. Jarman, A. Springer, W, Walker II, and W.G. Hunt. 1986. A metabolic derivation of DDE from kelthane. *Environmental Toxicology and Chemistry*. 5:13-19. (As cited in ATSDR, 2002).

Sabljic A. 1984. Predictions of the nature and strength of soil sorption of organic pollutants by molecular topology. *Journal of Agricultural and Food Chemistry*. 32:243-246. (As cited in ATSDR, 2002).

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

United States Environmental Protection Agency (USEPA). 1972. "DDT Ban Takes Effect." Press Release, December 31, 1972. Available on the Internet at: http://www.epa.gov/history/topics/ddt/01.htm. Accessed August 24, 2004. Last updated June 11, 2002.

USEPA. 1988. "Integrated Risk Information System (IRIS), p,p''-Dichlorodiphenyldichloroethylene (DDE)." August 1988. Available on the Internet at: http://www.epa.gov/iris/subst/0328.htm. Accessed February 2, 2005.

USEPA. 1990. *National Pesticide Survey: Survey Analytes.* EPA Report 570-9-90-NPS2. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS2.]

USEPA. 1992. *Pesticides in Ground Water Database: A Compilation of Monitoring Studies, 1971-1991. National Summary.* Office of Prevention, Pesticides and Toxic Substances. EPA Report 734-12-92-001. Available on the Internet at: http://nepis.epa.gov/pubtitleOPPTS.htm. [Search for document number 7341292001.]

USEPA. 1995a. *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1995b. Method 508 - Determination of Chlorinated Pesticides in Water by Gas Chromatography with and Electron Capture Detector. Revision 3.1. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1995c. Method 508.1 - Determination of Chlorinated Pesticides, Herbicides, and Organohalides by Liquid-Solid Extraction and Electron Capture Chromatography. Revision 2.0. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1995d. Method 525.2 - Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry. Revision 2.0. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1996. Integrated Risk Information System (IRIS), *p,p'*-Dichlorodiphenyltrichloroethane (DDT). February 1996. Available on the Internet at: http://www.epa.gov/iris/subst/0147.htm. Accessed February 2, 2005.

USEPA. 1999. *Guidelines for Carcinogen Risk Assessment.* NCEA-F-0644, Review Draft. July 1999. Available on the Internet at: http://www.epa.gov/iris/cancer_gls.pdf.

USEPA. 2000. *Unregulated Contaminant Monitoring Regulation Analytical Methods and Quality Control Manual*. EPA Report 815-R-00-006.

USEPA. 2001. *The Incorporation of Water Treatment Effects on Pesticide Removal and Transformations in Food Quality Protection Act (FQPA) Drinking Water Assessments*. Available on the Internet at: http://www.epa.gov/oppfead1/trac/science/water_treatment_bak.pdf.

USEPA. 2006. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

United States Geological Survey (USGS). 2000. *Pesticides in Stream Sediment and Aquatic Biota*. USGS Fact Sheet FS-092-00. 4 pp. Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/fs09200/fs09200.pdf.

Chapter 6:
1,3-Dichloropropene

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

1,3-Dichloropropene (1,3-DCP), a synthetic volatile organic compound (SVOC), is used as a pre-plant soil fumigant to control nematodes and other pests in soils to be planted with all types of food and feed crops. 1,3-DCP is typically injected 12 to 18 inches beneath the soil surface and can only be used by certified handlers. To mitigate risks to drinking water, 1999 labeling requirements restrict the use of 1,3-DCP in areas with shallow ground water and vulnerable soils in certain northern tier States; in fields within 100 feet of a drinking water well; and in areas overlying karst geology.

1,3-DCP is toxic to organs involved in metabolism (e.g., the liver), excretion of conjugated metabolites (e.g., urinary bladder and the kidney), and organs along the portals of entry (e.g., forestomach for oral administration; mucous membrane of the nasal passage and lungs for inhalation exposure). Exposure to 1,3-DCP does not appear to cause adverse reproductive or developmental effects.

The weight of evidence suggests that 1,3-DCP is likely to be carcinogenic to humans. This characterization is supported by tumor observations in chronic animal bioassays for both inhalation and oral routes of exposure. Using an oral cancer slope factor of 1×10^{-1} $(mg/kg/day)^{-1}$, EPA calculated a health reference level (HRL) of 0.4 µg/L at the 10^{-6} cancer risk level.

Estimates of national annual use during the 1990s vary widely. Based on information from a 1991 data call-in and other sources, EPA estimates that approximately 23 million pounds of 1,3-DCP were used annually from 1990 to 1995. The National Center for Food and Agricultural Policy (NCFAP) estimates that approximately 40 million pounds were used annually around 1992 and approximately 35 million pounds were used annually around 1997. Toxic Release Inventory (TRI) data suggest that 1,3-dichloropropene industrial releases are dominated by air emissions, and generally declined between 1988 and 2003.

To evaluate the extent of 1,3-dichloropropene in drinking water, EPA included 1,3-DCP as an analyte in the Unregulated Contaminant Monitoring (UCM) Round 1 and UCM Round 2 surveys. The minimum reporting levels (MRLs) for UCM Round 1 ranged from 0.02 to 10 µg/L and the MRLs for UCM Round 2 ranged from 0.08 to 1 µg/L. EPA also analyzed for 1,3-DCP using the samples from the small systems that were included in the first Unregulated Contaminant Monitoring Regulation (UCMR 1) survey. The MRL used for the UCMR 1 survey was 0.5 µg/L. Because some of these reporting limits exceeded the thresholds of interest, the occurrence analyses may result in an underestimate of the number of systems affected. However, the MRL values used for UCM Round 1 and UCM Round 2 as well as UCMR 1 are within the 10^{-4} to the 10^{-6} cancer risk range.

The UCM Round 1 Cross Section data indicate that approximately 0.16 percent (or 15) of the 9,164 public water systems (PWSs) sampled had detections of 1,3-DCP at concentrations greater than 0.2 µg/L (½ the HRL), affecting approximately 0.86 percent of the population served (or 438,000 of 51 million). The UCM Round 1 Cross Section data indicate that each one of those systems also had concentrations greater than 0.4 µg/L (the HRL). That is, 0.16 percent (or 15) of the 9,164 PWSs sampled had detections greater than 0.4 µg/L (the HRL), affecting

approximately 0.86 percent of the population served (or 438,000 of 51 million people). The 99th percentile of all detections was 2 µg/L and the maximum reported value was 2 µg/L.

The UCM Round 2 Cross Section data indicate that approximately 0.30 percent (or 50) of the 16,787 PWSs sampled had detections of 1,3-DCP at concentrations greater than ½ the HRL (0.2 µg/L), affecting approximately 0.42 percent of the population served (or 193,000 of 46 million). The UCM Round 2 Cross Section data indicate that approximately 0.23 percent (or 38) of the 16,787 PWSs sampled had detections of 1,3-DCP at concentrations greater than the HRL (0.4 µg/L), affecting approximately 0.33 percent of the population served (or 152,000 of 46 million). The 99th percentile of all detections was 39 µg/L and the maximum reported value was 39 µg/L.

Because the sample preservative used may have resulted in potential underestimates of occurrence for the UCM Rounds 1 and 2 data, EPA subsequently analyzed for 1,3-DCP using the samples provided by 796 of the small systems included in the recent UCMR 1 survey. None of the 3,719 samples from these 796 small systems (serving a total population of 2.8 million) had 1,3-DCP at concentrations of 0.5 µg/L or more (the minimum reporting limit used for the analysis of 1,3-DCP and a level that is slightly higher than the HRL).

EPA also evaluated several sources of supplemental information on 1,3-DCP occurrence in ambient water and drinking water, including the National Pesticide Survey, the Pesticides in Ground Water Database, a well water survey submitted by the pesticide registrant, the USGS VOC National Synthesis Random Source Water Survey and Focused Source Water Survey, and the National Highway Runoff Data and Methodology Synthesis.

The Agency has made a preliminary determination not to regulate 1,3-DCP with a national primary drinking water regulation (NPDWR). Because 1,3-DCP appears to occur infrequently at levels of health concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. EPA believes the 1999 pesticide labeling requirements, which are intended to mitigate risks to drinking water, may be one reason for the infrequent occurrence of 1,3-DCP at levels of concern in subsequent monitoring surveys.

EPA recognizes that 1,3-dichloropropene is listed as a probable human carcinogen. For this reason, the Agency encourages those States with public water systems that may have 1,3-dichloropropene in concentrations above the HRL to evaluate site-specific protective measures and to consider whether State-level action (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory document for 1,3-DCP to provide more recent health information. The updated Health Advisory will provide guidance for States with public water systems that may have 1,3-DCP at concentrations of will for above the HRL.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

a.i.	Active Ingredient
BMD	Benchmark Dose
CAAC	3-chloroacrylic acid
CAAL	3-chloroallyl alcohol
CAS	Chemical Abstracts Service
CCL	Contaminant Candidate List
CWS	Community Water System
1,3-DCP	1,3-Dichloropropene
ELCD	Electrolytic Conductivity Detector
GAC	Granular Activated Carbon
GC	Gas Chromatography
HRL	Health Reference Level
IRIS	Integrated Risk Information System
LOD	Limit of Detection
LOQ	Limit of Quantitation
MDL	Method Detection Limit
MRL	Minimum Reporting Level
MS	Mass Spectrometry
MTBE	Methyl tertiary-butyl ether
NAWQA	National Water Quality Assessment
NCFAP	National Center for Food and Agricultural Policy
NPDES	National Pollutant Discharge Elimination System
NPDWR	National Primary Drinking Water Regulation
NPS	National Pesticide Survey
NTIS	National Technical Information Service
NTP	National Toxicology Program
PGWDB	Pesticides in Ground Water Database
PID	Photoionization Detector
PWS	Public Water System
RfC	Reference Concentration
RfD	Reference Dose
TRI	Toxics Release Inventory
UCM	Unregulated Contaminant Monitoring
UCMR 1	First Unregulated Contaminant Monitoring Regulation
USGS	United States Geological Survey
VOC	Volatile Organic Compound

6 1,3-Dichloropropene

6.1 Definition

1,3-Dichloropropene is a volatile organic chemical (VOC) used as a pesticide. It is also known as 1,3-dichloropropylene or 1,3-DCP, and goes by the common trade names Telone II, Dedisol C, and Vorlex (HSDB, 2004). The Chemical Abstracts Service (CAS) registry number for 1,3-dichloropropene is 542-75-6. 1,3-Dichloropropene can exist in either *cis*- and *trans*-isomeric forms, and both forms are typically combined as a racemic mixture in commercial products (USEPA, 1998). The two isomers have very similar properties; thus, this report only treats them separately when appropriate.

6.1.1 Properties and Sources

1,3-Dichloropropene is a colorless to straw-colored or amber liquid with a pungent, sharp, sweet, irritating, chloroform-like odor (Ashford, 1994 as cited in HSDB, 2004; NIOSH, 2004). 1,3-Dichloropropene is used as a soil fumigant to control nematodes and other soil pests, particularly in the control of root predation (USEPA, 1998). Commercially, 1,3-dichloropropene is produced by Dow Agrosciences. It is miscible with hydrocarbons, halogenated solvents, esters and ketones (Tomlin, 1997 as cited in HSDB, 2004), and soluble in toluene, acetone, and octane (Lewis, 1997 as cited in HSDB, 2004). It can be synthesized by the dehydration of 1,3-dichloro-2-propanol, the dehydrochlorination of 1,2,3-trichloropropane, and the reaction of 3-chloro-2-propen-1-ol with phosphorous trichloride (Budavari, 1996 as cited in HSDB, 2004), as well as by chlorination of propylene (Sittig, 1980; Ashford, 1994 both as cited in HSDB, 2004) or allyl chloride (Gerhartz, 1985 as cited in HSDB, 2004). The physical and chemical properties of this VOC are summarized in Exhibit 6-1. The properties listed are common to both isomers except where noted.

Exhibit 6-1: Physical and Chemical Properties of 1,3-Dichloropropene

Identification	
CAS number	542-75-6
Molecular Formula	$C_3H_4Cl_2$
Physical and Chemical Properties	
Boiling Point	*cis*- isomer: 104 °C [1] *trans*- isomer: 112.6 °C [1]
Melting Point	< - 50 °C [2]
Molecular Weight	110.97 g/mol [3]
K_{oc}	20-42 L/kg [4]
Log K_{ow}	1.82 [2]
Water Solubility	*cis*- isomer: 2,180 mg/L at 25 °C [1] *trans*- isomer: 2,320 mg/L at 25 °C [1]
Vapor Pressure	*cis*- isomer: 34.3 mm Hg at 25 °C [1] *trans*- isomer 23.0 mm Hg at 25 °C [1]
Henry's Law Constant	3.55×10^{-3} atm-m^3/mole [5] 0.088 (dimensionless), predicted [6] 0.14 (dimensionless), from literature [6]
Freundlich Isotherm Constant (K)	200 (µg/g)(L/µg)$^{1/n}$ [7]

[1] *USEPA, 1998*

[2] *Tomlin, 1997 (as cited in HSDB, 2004)*

[3] *Budavari, 1996 (as cited in HSDB, 2004)*

[4] *derived from Speth et al., 2001*

[5] *Warner et al., 1987 (as cited in HSDB, 2004)*

[6] *Speth et al., 2001*

[7] *Gardner et al., 1990 (as cited in Speth et al., 2001)*

6.1.2 Environmental Fate and Behavior

1,3-Dichloropropene is applied to soil as a fumigant. It is estimated that 5-10 percent of the *cis* isomer is lost to the atmosphere from a warm moist sandy loam (USEPA, 1980 as cited in HSDB, 2004). The Henry's law constant and vapor pressure indicate that volatilization from moist and dry soil may be an important fate process (HSDB, 2004).

In soil, 1,3-dichloropropene can exist as a vapor or in solution. The phase has important mobility implications. In the vapor phase, 1,3-dichloropropene more strongly adsorbs to soil particles, and is of medium to low mobility in soil. The adsorption potential varies, however, with soil organic matter content and temperature. Adsorption increases with higher organic matter content and lower temperatures (Munnecke and Vangundy, 1979, Leistra, 1970, Swann *et al*., 1983, all as cited in ATSDR, 1992). The mobility of 1,3-dichloropropene in solution, on the

other hand, is relatively high because adsorption capacity is low in the aqueous phase. Consequently, aqueous 1,3-dichloropropene has the potential to leach to ground water (Swann *et al*., 1983 as cited in ATSDR, 1992). K_{oc} values for 1,3-dichloropropene (see Exhibit 6-1) also suggest high mobility in soil (HSDB, 2004). Data from a number of States and regions where 1,3-dichloropropene is used indicate that the compound does leach to ground water with normal agricultural use (USEPA, 1998).

Roberts and Stoydin (1976, as cited in HSDB, 2004) report a biodegradation half-life in soil of 3 to 4 weeks, although they speculate that some of the chemical may have been lost due to volatilization. Other researchers have reported half-lives for both isomers that range from 3 to 25 days (van der Pas and Leistra, 1987, Albrecht, 1987, both as cited in HSDB, 2004). The type of soil greatly affects the rate of biodegradation, with half-lives of 1.8, 12.3, and 61 days observed in aerobically incubated Wahiawa silt clay, Catlin silt loam, and Fuquay loamy sand, respectively (Batzer *et al*., 1997 as cited in HSDB, 2004).

1,3-Dichloropropene in soil is also subject to hydrolysis. Krijgsheld and van der Gen (1986 as cited in HSDB, 2004) have reported hydrolysis half-lives in soil of 1.5 to 20 days at 20 ^{o}C and 91 to 100 days at 2 ^{o}C. Hydrolysis of the *cis*- and *trans*- isomers results in the formation of the corresponding 3-chloroallyl alcohols, which then form the corresponding 3-chloroallylacrylic acids (Albrecht, 1987 as cited in HSDB, 2004).

The Henry's Law constant indicates that 1,3-dichloropropene is expected to volatilize from water (Lyman *et al*., 1990 as cited in HSDB, 2004). A half-life of less than five hours for the evaporation of 1,3-dichloropropene from ditch water samples has been reported (Yon *et al*., 1991 as cited in HSDB, 2004).

6.2 Health Effects

Chronic and subchronic exposures to 1,3-DCP at doses of 12.5 mg/kg/day and above in animal dietary studies indicate that 1,3-DCP is toxic to organs involved in metabolism (liver), excretion of conjugated metabolites (e.g., urinary bladder and the kidney) and organs along the portals of entry (e.g., forestomach for oral administration; mucous membrane of the nasal passage and lungs for inhalation exposure). Exposure to 1,3-DCP has not been shown to cause reproductive or developmental effects. Neither reproductive nor developmental toxicity were observed in a two-generation reproductive study in rats or in developmental studies in rats and rabbits at maternal inhalation concentrations up to 376 mg/m^3 (USEPA, 2000). Even concentrations that produced parental toxicity did not produce reproductive or developmental effects (USEPA, 2000).

A reference does (RfD) of 0.03 mg/kg/day for 1,3-DCP (USEPA, 2000) has been established using a benchmark dose (BMD) analysis based on a two-year chronic bioassay (Stott *et al.*, 1995 as cited in USEPA, 2000) in which chronic irritation (forestomach hyperplasia) and significant body weight reduction were the critical and co-critical effects, respectively. A reference concentration (RfC) of 0.02 mg/m^3 was derived from a two-year bioassay (Lomax *et al.*, 1989 as cited in USEPA, 2000), which observed histopathology in the nasal epithelium.

Under the proposed cancer risk assessment guidelines, the weight of evidence for evaluation of 1,3-DCP's ability to cause cancer suggests that it is likely to be carcinogenic to humans (USEPA, 2000). This characterization is supported by tumor observations in chronic animal bioassays for both inhalation and oral routes of exposure.

The oral cancer slope factors calculated from chronic dietary, gavage and inhalation data ranged from 5×10^{-2} to 1×10^{-1} $(mg/kg/day)^{-1}$. Due to uncertainties in the delivered doses in some studies, EPA's Integrated Risk Information System (IRIS) recommended using the oral slope factor of 1×10^{-1} $(mg/kg/day)^{-1}$ from a National Toxicology Program study (NTP, 1985). Using this oral slope factor, EPA calculated a health reference level (HRL) of 0.4 µg/L at the 10^{-6} cancer risk level.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. No human or animal studies are available that have examined the effect of 1,3-DCP exposure on juvenile subjects. Therefore, its effects on children are unknown. Developmental studies in rats and rabbits show no evidence of develop-mental effects and therefore it is unlikely that 1,3-DCP causes developmental toxicity.

6.3 Occurrence and Exposure

6.3.1 Use and Environmental Release

1,3-Dichloropropene, marketed under the trade name "Telone," is used as a soil fumigant to control nematodes and other soil pests. It is applied before planting, and generally injected 12 to 18 inches into the soil to minimize volatilization. 1,3-Dichloropropene was first registered for use in the United States in 1954. It is currently registered for commercial cultivation of all types of food and feed crops, including vegetable, fruit and nut crops, forage crops (grasses, legumes and other non-grass forage crops), tobacco, fiber crops, and nursery crops (ornamental, non-bearing fruit/nut trees and forestry crops). 1,3-Dichloropropene can only be applied by certified operators; it is not registered for household use. Since 1999, use of 1,3-dichloropropene has been restricted to mitigate risks to ground water. Use of the fumigant is prohibited within 100 feet of drinking water wells, in areas overlying karst geology, and in parts of certain northern tier States (ND, SD, WI, MN, NY, ME, NH, VT, MA, UT, MT) where aquifers are shallow and soils are porous (USEPA, 1998).

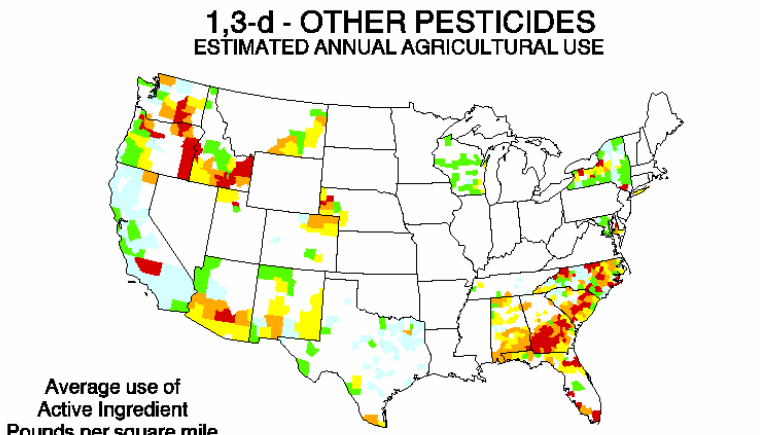
National use estimates are available. Using data from a variety of published sources and its own proprietary data, mostly from a 1991 data call-in, USEPA (1998) estimated that approximately 23 million pounds of active ingredient (a.i.) were used annually to treat approximately 372 thousand acres during the years 1990-1995. The National Center for Food and Agricultural Policy (NCFAP) lists uses of 1,3-dichloropropene on 17 crops totaling approximately 40.1 million pounds a.i. per year in 1992, and uses on 18 crops totaling approximately 34.7 million pounds of a.i. per year in 1997 (NCFAP, 2003). For more information on NCFAP pesticide estimates, see Chapter 2.

The United States Geological Survey (USGS) combined data collected by NCFAP with data from the Census of Agriculture to estimate that 40.0 million pounds of 1,3-dichloropropene a.i. per year were used in agriculture in the early 1990s (Thelin and Gianessi, 2000). While

USGS has not published national estimates for 1997, an estimate of approximately 33.5 million pounds a.i. can be inferred from the "total pounds applied" and "percent national use" data in the 1997 geographical distribution map (Exhibit 6-2).

Exhibit 6-2 shows the estimated geographic distribution and intensity of typical annual 1,3-dichloropropene use in the United States in the late 1990s. A breakdown of use by crop is also included. The map was created by USGS using State-level data sets on pesticide use rates from 1995-1998 compiled by NCFAP, combined with county-level data on harvested crop acreage obtained from the 1997 Census of Agriculture (USGS, 2004). Due to the nature of the data sources, non-agricultural uses are not reflected here and variations in use at the county-level are also not well represented (Thelin and Gianessi, 2000). However, because there are no registered residential uses for 1,3-dichloropropene, non-agricultural use is expected to be insignificant (USEPA, 1998). For more background on the USGS pesticide use maps, see Chapter 2. The map indicates that 1,3-dichloropropene use is concentrated in the Southeast, the Southwest, and the Northwest of the country, with isolated pockets elsewhere.

Exhibit 6-2: Estimated Annual Agricultural Use of 1,3-Dichloropropene (c. 1997)



Crops	Total Pounds Applied	Percent National Use
tobacco	13,197,891	39.38
potatoes	9,805,178	29.26
cotton	3,610,261	10.77
onions	1,797,829	5.36
carrots	1,510,079	4.51
hot peppers	939,618	2.79
sugarbeets	718,848	2.14
sweet potatoes	697,364	2.08
peanuts	503,883	1.50
strawberries	174,785	0.52

Source: USGS, 2004

1,3-Dichloropropene is listed as a Toxics Release Inventory (TRI) chemical. For a discussion of the nature and limitations of TRI data, see Chapter 2.

TRI data for 1,3-dichloropropene (see Exhibit 6-3) are reported for the years 1988 to 2003 (USEPA, 2006a). Air emissions constitute most of the on-site releases (and total releases), and generally decrease throughout the period of record. A sharp decline is evident between 1995 and 1996, and a modest increase in 2000 and 2001. Surface water discharges are of secondary importance, and no obvious trend is evident. Reported underground injection, releases to land, and off-site releases are generally insignificant. TRI releases of 1,3-dichloropropene were reported from facilities in 17 States (AR, CA, DE, FL, GA, HI, IL, KY, LA, MI, MS, NJ, NC, OH, SC, TX, and WA), although not all States had facilities reporting releases every year.

Exhibit 6-3: Environmental Releases (in pounds) of 1,3-Dichloropropene in the United States, 1988-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1988	54,590	250	0	0	0	54,840
1989	50,917	340	0	0	3,354	54,611
1990	59,473	310	0	0	0	59,783
1991	20,405	0	0	0	0	20,405
1992	37,711	69	0	0	0	37,780
1993	33,348	2	0	0	0	33,350
1994	24,670	86	0	0	0	24,756
1995	32,977	193	0	0	0	33,170
1996	10,875	1,270	0	0	0	12,145
1997	10,131	67	0	0	0	10,198
1998	11,566	61	0	1	0	11,628
1999	6,600	68	0	0	168	6,836
2000	10,295	288	2	200	10	10,795
2001	13,062	460	0	0	505	14,027
2002	9,860	85	0	332	255	10,532
2003	8,256	6	0	412	250	8,924

Source: USEPA, 2006a

6.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are sources of drinking water. Recent data on the occurrence of 1,3-dichloropropene in ambient surface and ground water are available from the National Water Quality Assessment (NAWQA) program of the USGS. For details on this program, see the discussion in Chapter 2. USGS has also collected data on 1,3-dichloropropene occurrence in reviews of existing literature.

NAWQA VOC National Synthesis

Random and Focused VOC Surveys

Using data collected from the NAWQA study units and other sources, USGS and collaborating institutions have recently completed a national assessment of VOC occurrence in the nation's drinking water supply. The assessment included a random survey (1999-2000) of VOC occurrence in ground and surface water resources used by geographically representative community water systems (CWSs) in different size categories (Grady, 2003) and a focused survey (1999-2001) of VOC occurrence patterns, including seasonal variability, in source waters considered particularly susceptible to methyl tertiary butyl ether (MTBE) contamination (Delzer and Ivahnenko, 2003). 1,3-Dichloropropene was included as an analyte in both surveys, with a reporting level of 0.2 µg/L (Ivahnenko *et al.*, 2001).

Neither the national random survey nor the focused survey found any detections of 1,3-dichloropropene at the reporting level of 0.2 µg/L (Grady, 2003; Delzer and Ivahnenko, 2003).

Even when evaluating occurrence at levels as low as the method detection limit (0.024 µg/L for *cis*-1,3-dichloropropene and 0.026 µg/L for *trans*-1,3-dichloropropene), the focused survey found no detections of either isomer (Delzer and Ivahnenko, 2003).

Compilation of Historical VOC Monitoring Data

USGS (Squillace *et al.*, 1999) assessed VOC occurrence in untreated ambient ground water samples collected between 1985 and 1995 by local, State, and federal agencies. The samples represented both urban and rural areas, and both drinking water and non-drinking water wells.

Multiple investigators collected *cis*-1,3-dichloropropene samples from 349 urban wells and 2,138 rural wells and *trans*-1,3-dichloropropene samples from 347 urban wells and 2,039 rural wells. At a reporting level of 0.2 µg/L, there were no detections of either isomer (Squillace *et al.*, 1999).

USGS Stormwater Studies

For the National Highway Runoff Data and Methodology Synthesis, USGS conducted a review of 44 highway and urban runoff studies implemented since 1970 (Lopes and Dionne, 1998). 1,3-Dichloropropene results are reported in four of these studies. For more information on this collection of studies, see Chapter 2.

Three of the studies were stormwater studies conducted in major metropolitan areas in connection with National Pollutant Discharge Elimination System (NPDES) permitting. In metropolitan Phoenix (Maricopa County), USGS collected 35 samples from five drainage basins and the City of Phoenix collected an additional 26 samples from seven sites (Lopes *et al.*, 1995). In Colorado Springs, 35 samples were collected from five sites (von Guerard and Weiss, 1995). In Dallas-Fort Worth, 182 samples were collected from 26 stormwater drainage basins (Baldys *et al.*, 1998). The reporting limits were 0.2 µg/L in Phoenix and Colorado Springs, and they ranged from 0.2 to 10 µg/L in Dallas/Fort Worth. Not all samples were monitored for every contaminant. These three studies reported no detections of 1,3-dichloropropene.

The fourth study analyzed 86 urban runoff samples from 15 cities, collected between 1979 and 1982 in connection with the National Urban Runoff Program (Cole *et al.*, 1984). 1,3-Dichloropropene was detected in 2 percent of samples, in concentrations ranging from 1 µg/L to 2 µg/L. All detections were from Eugene, Oregon. A detection limit was not reported.

6.3.2 Drinking Water Occurrence

Nationally representative data on 1,3-dichloropropene occurrence in drinking water were collected by large and small public water systems under EPA's Unregulated Contaminant Monitoring (UCM) program (1987-1999). However, there are doubts about the reliability of these data. Subsequently, additional 1,3-dichloropropene monitoring has been conducted, using a revised protocol, in conjunction with recent Unregulated Contaminant Monitoring Regulation 1 (UCMR 1) monitoring.

UCM Program, Rounds 1 and 2

1,3-Dichoropropene monitoring results from UCM Rounds 1 and 2 may have been compromised by the widespread use of sodium sulfate and sodium thiosulfate as dechlorinating agents. Before it was recognized that sodium sulfate and sodium thiosulfate degrade 1,3-dichloropropene in analytical samples, the two compounds were commonly used to preserve drinking water samples for VOC testing. Hence, older drinking water surveys like UCM Rounds 1 and 2 likely underestimate actual 1,3-dichloropropene occurrence. (This concern does not apply to the ambient 1,3-dichloropropene monitoring described above. USGS's ambient monitoring typically does not involve a dechlorination step. In rare cases when dechlorination is necessary, USGS employs ascorbic acid as the dechlorinating agent.)

With the caveat that UCM occurrence estimates are likely underestimates, it is still instructive to analyze the occurrence data collected. Round 1 of the UCM lasted from 1988 to 1992, and Round 2 lasted from 1993 to 1999. A geographical cross-section of States with the most complete and reliable data was chosen to provide a roughly representative picture of national occurrence in each round. For details on the UCM program, see Chapter 2 and USEPA (2006b).

Exhibits 6-4 and 6-5 show the results from the Round 1 and Round 2 cross-sections. Results from all States, including those with incomplete and less reliable data, are also presented for the sake of comparison. Results are analyzed at the level of simple detections (at or above the minimum reporting level, or ≥ MRL), exceedances of the health reference level (> HRL, or > 0.4 µg/L), and exceedances of one half the value of the HRL (> ½ HRL, or > 0.2 µg/L). MRLs for 1,3-dichloropropene were not uniform. They varied from 0.02 to 10 µg/L in the first Round, and from 0.08 to 1 µg/L in the second Round. The modal (most common) MRL in both Rounds was 0.5 µg/L. Because the MRL was often higher than the HRL and ½ HRL, it is likely that the sampling failed to capture some ½ HRL and HRL exceedances at the participating systems, and that the ½ HRL and HRL analyses underestimate actual 1,3-dichloropropene occurrence. However, all MRLs fell within (or below) the risk range of 10^{-6} to 10^{-4} used by EPA to evaluate carcinogens (see Section 2.1.1).

In Round 1 cross-section States, 1,3-dichloropropene was detected at approximately 0.16% of public water systems (PWSs), affecting 0.86% of the population served, equivalent to approximately 1.8 million people nationally. All of these detections were at concentrations higher than the HRL. This is not surprising, since the most common MRL, 0.5 µg/L, is higher than the HRL.

When all Round 1 results are included in the analysis, including results from States with incomplete or less reliable data, 1,3-dichloropropene detection frequencies appear to be slightly higher than the cross-section data indicate. Detections affect 0.20% of PWSs and 0.95% of the population served; exceedances of the HRL (and ½ HRL) affect 0.19% of PWSs and 0.94% of the population served.

In Round 2 cross-section States, 1,3-dichloropropene was detected at 0.35% of PWSs, affecting 0.55% of the population served, equivalent to approximately 1.2 million people nationally. The ½ HRL benchmark was exceeded in 0.30% of PWSs, affecting 0.42% of the

population served, equivalent to approximately 0.9 million people nationally. The HRL benchmark was exceeded in 0.23% of PWSs, affecting 0.33% of the population served, equivalent to approximately 0.7 million people nationally. Compared with Round 1, Round 2 shows greater occurrence of 1,3-dichloropropene across the board, and shows a greater proportion of detections at low levels that do not exceed the health-related benchmarks. Both of these phenomena are at least partly explained by the fact that the analytical detection methods used in Round 2 were generally more sensitive.

When all Round 2 results are included in the analysis, 1,3-dichloropropene occurrence findings appear to be slightly lower than those observed for the cross-section data. Detections affect 0.31% of PWSs and 0.47% of the population served; ½ HRL exceedances affect 0.27% of PWSs and 0.36% of the population served; and HRL exceedances affect 0.20% of PWSs and 0.27% of the population served.

Exhibit 6-4: Summary UCM Occurrence Statistics for 1,3-Dichloropropene (Round 1)

Frequency Factors	24-State Cross-Section[1]		All Reporting States[2]		National System & Population Numbers[3]	
Total Number of Samples	31,104		31,973		--	
Percent of Samples with Detections	0.06%		0.09%		--	
99th Percentile Concentration (all samples)	< MRL		< MRL		--	
Health Reference Level (HRL)	0.4 µg/L		0.4 µg/L		--	
Minimum Reporting Level (MRL) - Range - (modal value)[4]	0.02 - 10 µg/L (0.5 µg/L)		0.02 - 10 µg/L (0.5 µg/L)		--	
Maximum Concentration of Detections	2.0 µg/L		17.0 µg/L		--	
99th Percentile Concentration of Detections	2.0 µg/L		15.6 µg/L		--	
Median Concentration of Detections	1.0 µg/L		1.0 µg/L		--	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	9,164 8,303 898		9,307 8,401 947		65,030 59,440 5,590	
Total Population Population of GW PWSs Population of SW PWSs	50,917,006 24,660,968 29,271,833		52,879,061 26,106,876 29,867,090		213,008,182 85,681,696 127,326,486	
Occurrence by System	**Number**	**Percentage**	**Number**	**Percentage**	**National Extrapolation[5]**	
					Cross-Section	**All States**
PWSs with detections (≥ MRL) Range across States GW PWSs with detections SW PWSs with detections	15 0 - 7 10 5	0.16% 0 - 1.75% 0.12% 0.56%	19 0 - 7 14 6	0.20% 0 - 100% 0.17% 0.63%	106 N/A 72 31	133 N/A 99 35
PWSs > 1/2 HRL Range across States GW PWSs > 1/2 HRL SW PWSs > 1/2 HRL	15 0 - 7 10 5	0.16% 0 - 1.75% 0.12% 0.56%	18 0 - 7 13 6	0.19% 0 - 100% 0.15% 0.63%	106 N/A 72 31	126 N/A 92 35
PWSs > HRL Range across States GW PWSs > HRL SW PWSs > HRL	15 0 - 7 10 5	0.16% 0 - 1.75% 0.12% 0.56%	18 0 - 7 13 6	0.19% 0 - 100% 0.15% 0.63%	106 N/A 72 31	126 N/A 92 35
Occurrence by Population Served						
Population served by PWSs with detections Range across States Pop. Served by GW PWSs with detections Pop. Served by SW PWSs with detections	436,223 0 - 225,630 146,155 290,068	0.86% 0 - 6.12% 0.59% 0.99%	500,486 0 - 225,630 210,418 342,118	0.95% 0 - 100% 0.81% 1.15%	1,825,000 N/A 508,000 1,262,000	2,016,000 N/A 691,000 1,458,000
Population served by PWSs > 1/2 HRL Range across States Pop. Served by GW PWSs > 1/2 HRL Pop. Served by SW PWSs > 1/2 HRL	436,223 0 - 225,630 146,155 290,068	0.86% 0 - 6.12% 0.59% 0.99%	497,246 0 - 225,630 207,178 342,118	0.94% 0 - 100% 0.79% 1.15%	1,825,000 N/A 508,000 1,262,000	2,003,000 N/A 680,000 1,458,000
Population served by PWSs > HRL Range across States Pop. Served by GW PWSs > HRL Pop. Served by SW PWSs > HRL	436,223 0 - 225,630 146,155 290,068	0.86% 0 - 6.12% 0.59% 0.99%	497,246 0 - 225,630 207,178 342,118	0.94% 0 - 100% 0.79% 1.15%	1,825,000 N/A 508,000 1,262,000	2,003,000 N/A 680,000 1,458,000

1. Summary Results based on 24-State Cross-Section, UCM Round 1 data.
2. Summary Results based on All Reporting States, UCM Round 1 data.
3. Total PWS and population numbers are from EPA March 2000 Water Industry Baseline Handbook, 2nd Edition.
4. Because several different analytical methods were used, MRLs were not uniform. The modal value is the most common MRL.
5. National extrapolations are generated by multiplying the system/population percentages and the national Baseline Handbook system/population numbers.

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with Detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with Detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-Because some systems were counted as both ground water and surface water systems and others could not be classified, GW and SW figures might not add up to totals.
-Due to differences between the ratios of GW and SW systems with monitoring results and the national ratio, extrapolated GW and SW figures might not add up to extrapolated totals.
-Due to MRL variability, it is likely that the sampling failed to capture some ½ HRL and HRL exceedances at the participating systems, and the ½ HRL and HRL analyses underestimate actual contaminant occurrence.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 6-5: Summary UCM Occurrence Statistics for 1,3-Dichloropropene (Round 2)

Frequency Factors	20-State Cross-Section[1]		All Reporting States[2]		National System & Population Numbers[3]	
Total Number of Samples	70,631		79,388		--	
Percent of Samples with Detections	0.11%		0.10%		--	
99th Percentile Concentration (all samples)	< MRL		< MRL		--	
Health Reference Level (HRL)	0.4 µg/L		0.4 µg/L		--	
Minimum Reporting Level (MRL) - Range - (modal value)[4]	0.08 - 1 µg/L (0.5 µg/L)		0.08 - 1 µg/L (0.5 µg/L)		--	
Maximum Concentration of Detections	39 µg/L		39 µg/L		--	
99th Percentile Concentration of Detections	39 µg/L		25 µg/L		--	
Median Concentration of Detections	0.5 µg/L		0.5 µg/L		--	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	16,787 15,178 1,609		18,944 17,098 1,846		65,030 59,440 5,590	
Total Population Population of GW PWSs Population of SW PWSs	45,951,052 17,423,030 28,528,022		55,713,623 21,446,615 34,267,008		213,008,182 85,681,696 127,326,486	
Occurrence by System	**Number**	**Percentage**	**Number**	**Percentage**	**National Extrapolation[5]**	
					Cross-Section	**All States**
PWSs with detections (≥ MRL) Range across States GW PWSs with detections SW PWSs with detections	58 0 - 43 48 10	0.35% 0 - 2.91% 0.32% 0.62%	59 0 - 43 48 11	0.31% 0 - 2.91% 0.28% 0.60%	225 N/A 188 35	203 N/A 167 33
PWSs > 1/2 HRL Range across States GW PWSs > 1/2 HRL SW PWSs > 1/2 HRL	50 0 - 35 41 9	0.30% 0 - 2.36% 0.27% 0.56%	51 0 - 35 41 10	0.27% 0 - 2.36% 0.24% 0.54%	194 N/A 161 31	175 N/A 143 30
PWSs > HRL Range across States GW PWSs > HRL SW PWSs > HRL	38 0 - 23 29 9	0.23% 0 - 1.55% 0.19% 0.56%	38 0 - 23 29 9	0.20% 0 - 1.55% 0.17% 0.49%	147 N/A 114 31	130 N/A 101 27
Occurrence by Population Served						
Population served by PWSs with detections Range across States Pop. Served by GW PWSs with detections Pop. Served by SW PWSs with detections	252,643 0 - 209,261 197,066 55,577	0.55% 0 - 5.78% 1.13% 0.19%	260,157 0 - 209,261 197,066 63,091	0.47% 0 - 5.78% 0.92% 0.18%	1,171,000 N/A 969,000 248,000	995,000 N/A 787,000 234,000
Population served by PWSs > 1/2 HRL Range across States Pop. Served by GW PWSs > 1/2 HRL Pop. Served by SW PWSs > 1/2 HRL	192,870 0 - 149,488 141,275 51,595	0.42% 0 - 4.13% 0.81% 0.18%	200,384 0 - 149,488 141,275 59,109	0.36% 0 - 4.13% 0.66% 0.17%	894,000 N/A 695,000 230,000	766,000 N/A 564,000 220,000
Population served by PWSs > HRL Range across States Pop. Served by GW PWSs > HRL Pop. Served by SW PWSs > HRL	151,553 0 - 108,171 99,958 51,595	0.33% 0 - 2.99% 0.57% 0.18%	151,553 0 - 108,171 99,958 51,595	0.27% 0 - 2.99% 0.47% 0.15%	703,000 N/A 492,000 230,000	579,000 N/A 399,000 192,000

1. *Summary Results based on 20-State Cross-Section, UCM Round 2 data.*
2. *Summary Results based on All Reporting States, UCM Round 2 data.*
3. *Total PWS and population numbers are from EPA March 2000 Water Industry Baseline Handbook, 2nd Edition.*
4. *Because several different analytical methods were used, MRLs were not uniform. The modal value is the most common MRL.*
5. *National extrapolations are generated by multiplying the system/population percentages and the national Baseline Handbook system/population numbers.*

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with Detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with Detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-Due to differences between the ratios of GW and SW systems with monitoring results and the national ratio, extrapolated GW and SW figures might not add up to extrapolated totals.
-Due to MRL variability, it is likely that the sampling failed to capture some ½ HRL and HRL exceedances at the participating systems, and the ½ HRL and HRL analyses underestimate actual contaminant occurrence.
-The HRL used in this analysis is a draft value for working review only.

Each of the following maps focuses on a somewhat different aspect of the geographical distribution of 1,3-dichloropropene occurrence. The first exhibit (Exhibit 6-6) identifies all States with at least one PWS with a detection of 1,3-dichloropropene in Round 1 or Round 2. All States are included in this analysis, including both cross-section States with reliable data and non-cross-section States with less reliable data, in order to provide the broadest assessment of possible 1,3-dichloropropene occurrence. The second exhibit (Exhibit 6-7) presents the same information (identifying States with detections, regardless of whether they were included in the cross-sections) separately for Round 1 (1988-1992) and Round 2 (1993-1999), to reveal temporal trends.

The third exhibit (Exhibit 6-8) illustrates the geographic distribution of States with different detection frequencies (percentage of PWSs with at least one detection), and the fourth exhibit (Exhibit 6-9) illustrates the geographic distribution of different HRL exceedance frequencies (percentage of PWSs with at least one HRL exceedance). Only cross-section States, which have the most complete and reliable occurrence data, are included in these two analyses. In each exhibit, Round 1 data are presented in the upper map and Round 2 data are presented in the lower map to reveal temporal trends.

In each map, two color categories represent States with no data. States in white do not belong to the relevant Round or cross-section, and States in the lightest category of shading were included in the Round or cross-section but have no data for 1,3-dichloropropene. The darker shades are used to differentiate occurrence findings in States with 1,3-dichloropropene data.

These maps reveal no clear geographic or temporal patterns of 1,3-dichloropropene occurrence. States with PWSs with detections are distributed from the east to the west coast, and from the Canadian to the Mexican borders. Even the States with the highest proportion of PWSs with detections are generally distributed across the United States.

Exhibit 6-6: Geographic Distribution of 1,3-Dichloropropene Detections in Both Cross-Section and Non-Cross-Section States (Combined UCM Rounds 1 and 2)

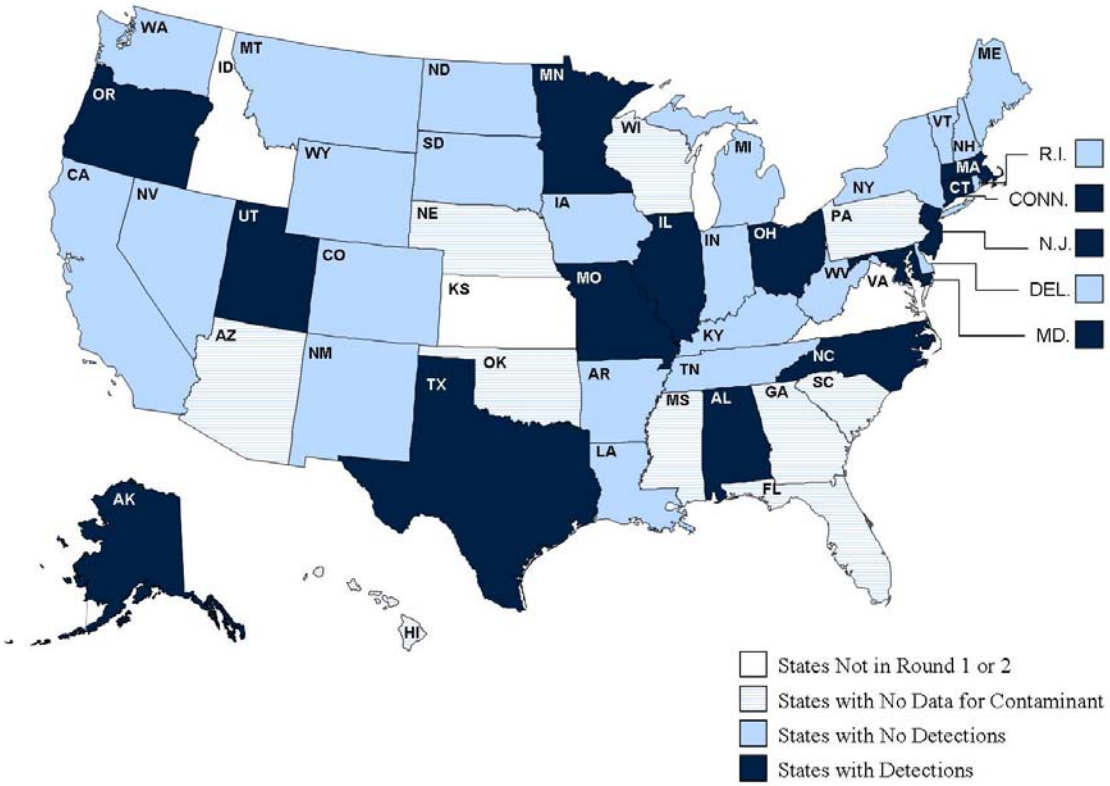
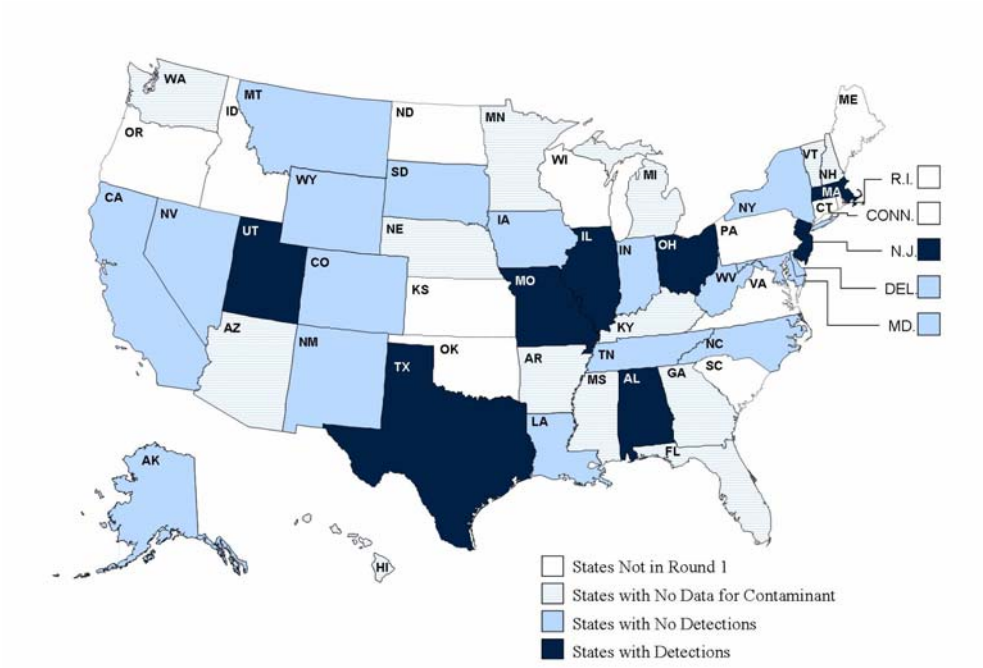


Exhibit 6-7: Geographic Distribution of 1,3-Dichloropropene Detections in Both Cross-Section and Non-Cross-Section States (Above: UCM Round 1; Below: UCM Round 2)



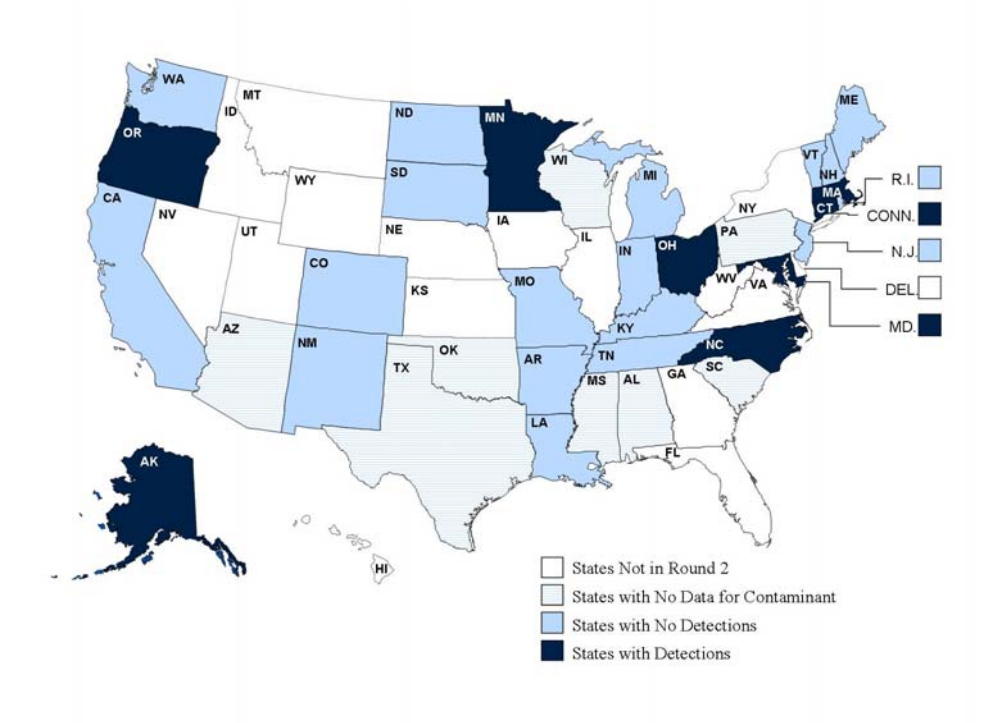
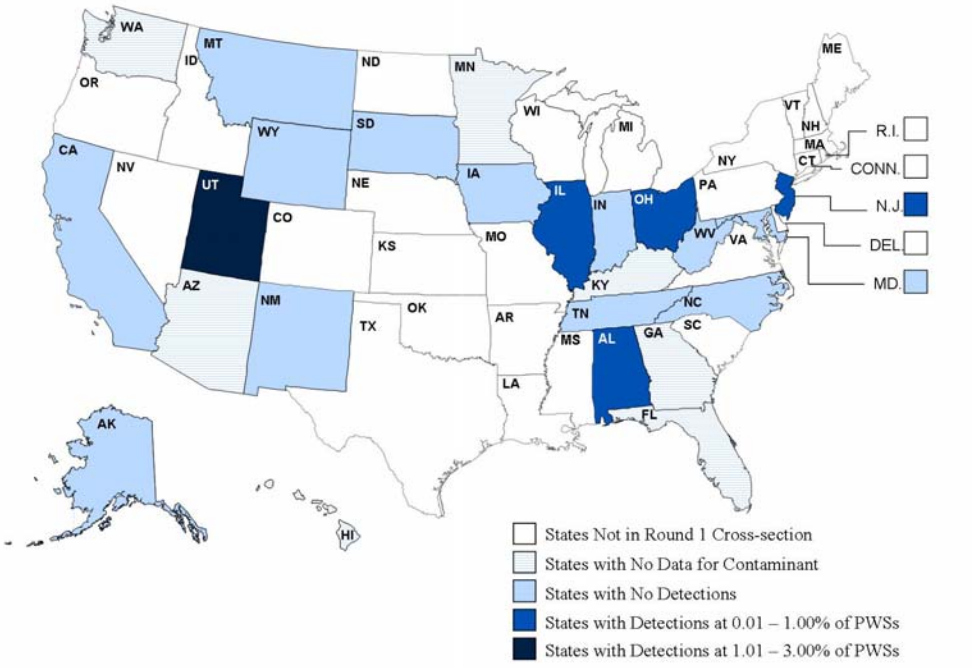


Exhibit 6-8: Geographic Distribution of 1,3-Dichloropropene Detection Frequencies in Cross-Section States (Above: UCM Round 1; Below: UCM Round 2)



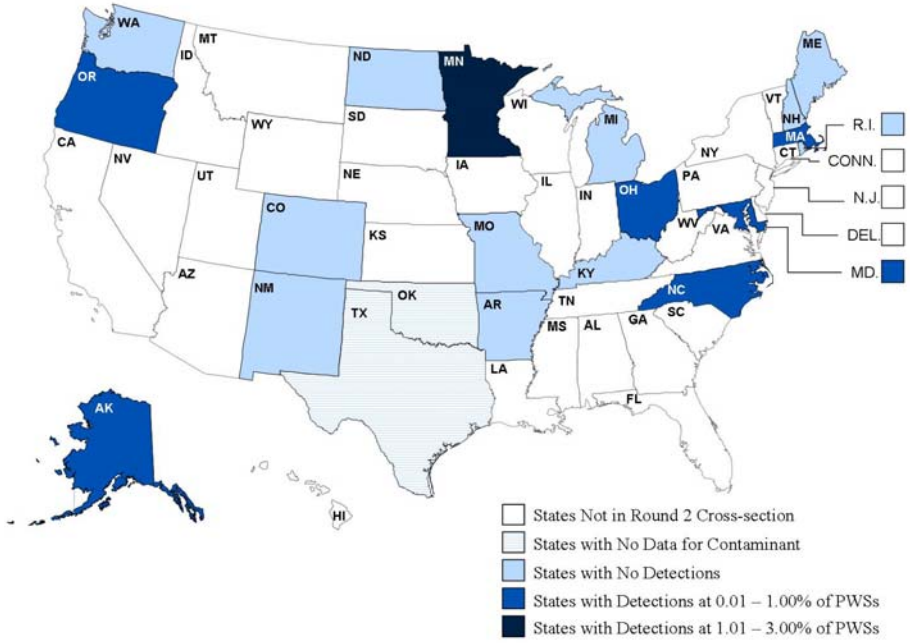
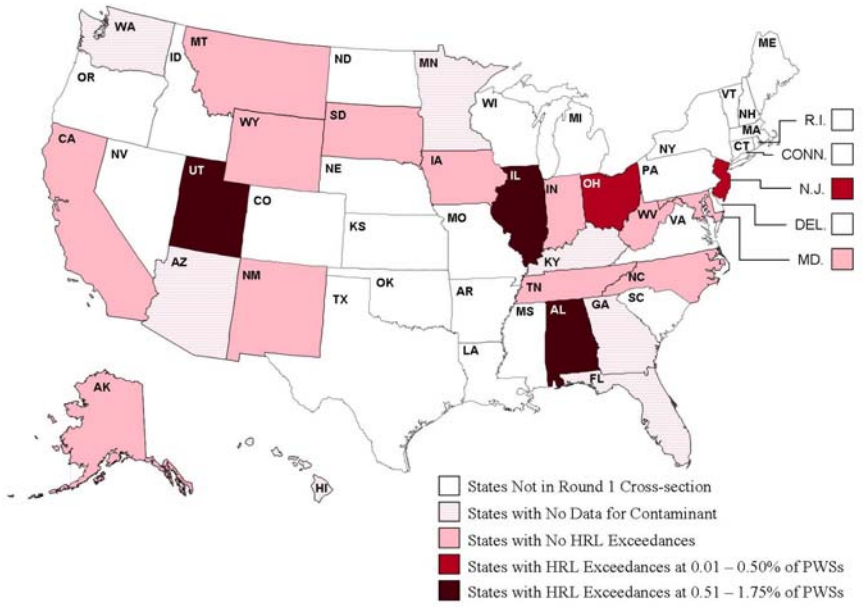
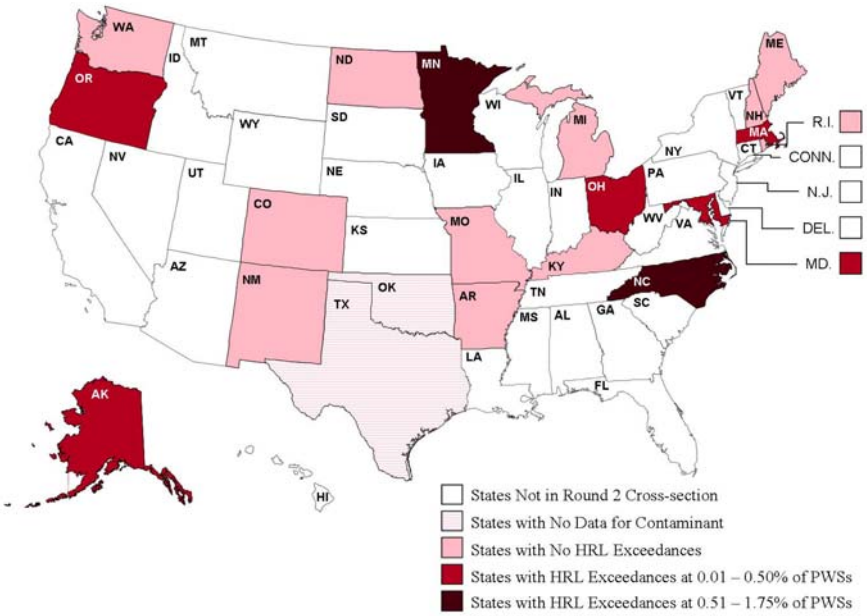


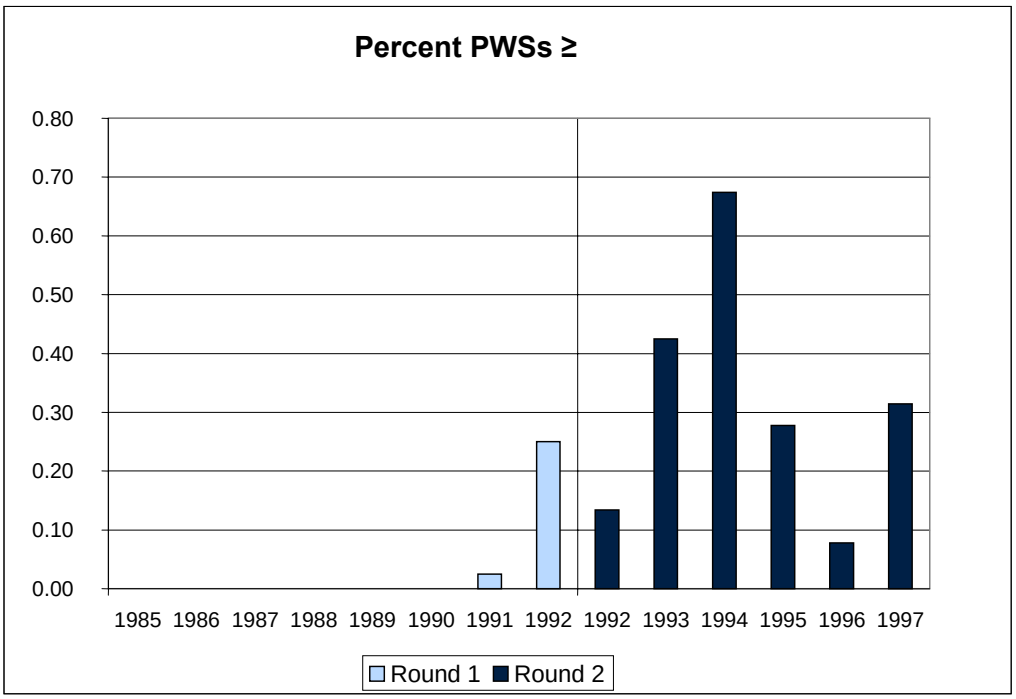
Exhibit 6-9: Geographic Distribution of 1,3-Dichloropropene HRL Exceedance Frequencies in Cross-Section States (Above: UCM Round 1; Below: UCM Round 2)

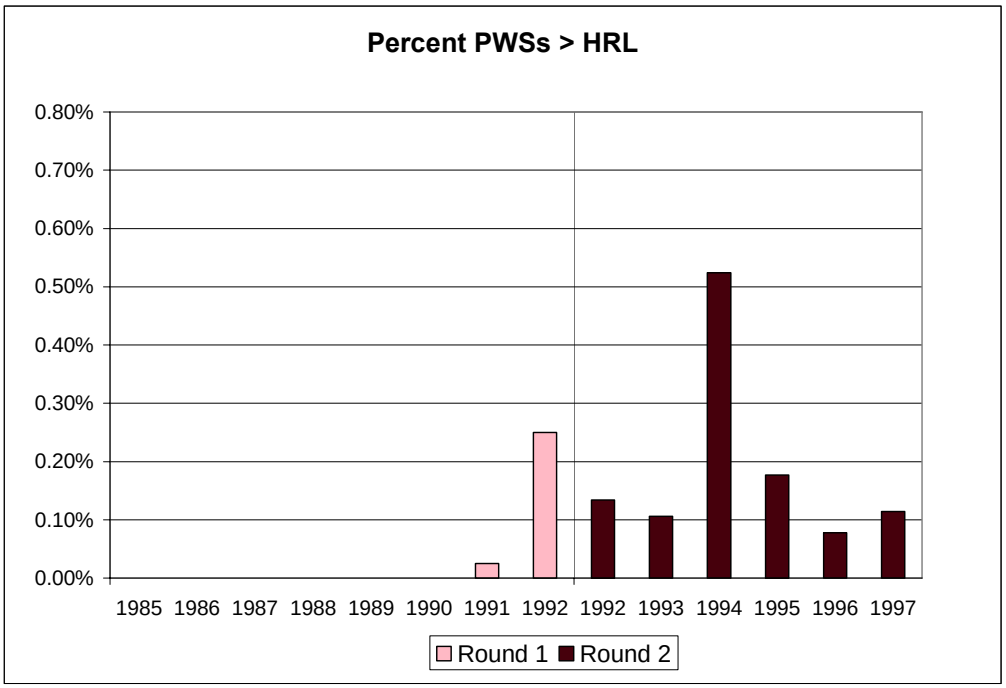




Eight States (AK, KY, MD, MN, NC, NM, OH, and WA) contributed 1,3-dichloropropene data to both the Round 1 and Round 2 cross-sections. While these States are not necessarily nationally representative, they enable a preliminary assessment of temporal trends in 1,3-dichloropropene occurrence. Exhibits 6-10 and 6-11 indicate that both detections and HRL exceedances began in 1991 and peaked in 1994, and that by far the State with the highest rate of detections, among the eight, was Minnesota.

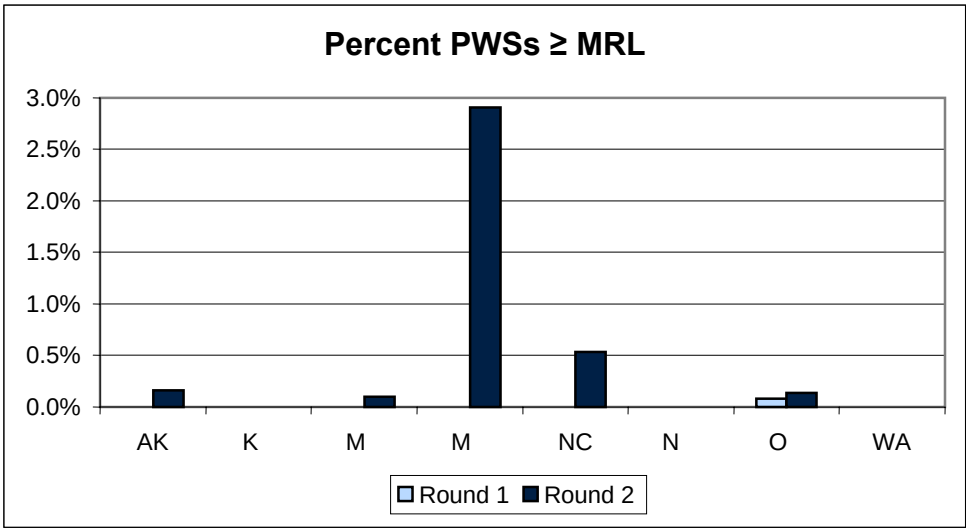
Exhibit 6-10: Annual Frequency of 1,3-Dichloropropene Detections (above) and HRL Exceedances (below), 1985 - 1997, in Select Cross-Section States

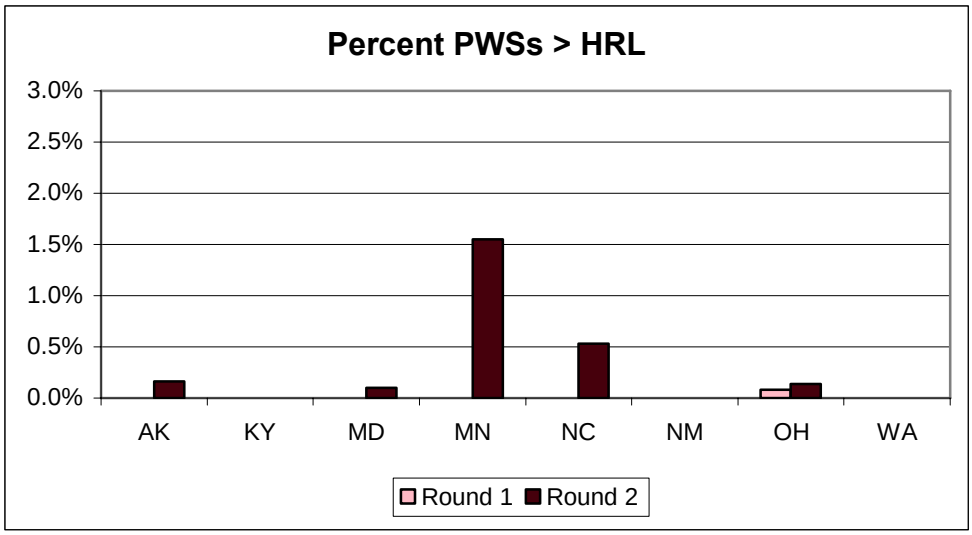




Note: Data are from AK, KY, MD, MN, NC, NM, OH, and WA. (These eight States are the only States in both the Round 1 and the Round 2 cross-sections.) Both Round 1 and Round 2 have data for 1992; 1992 results from each Round are presented separately. The HRL for 1,3-dichloropropene is 0.4 µg/L.

Exhibit 6-11: Distribution of 1,3-Dichloropropene Detections (above) and HRL Exceedances (below) Among Select Cross-Section States





Note: These eight States are the only States in both the Round 1 and Round 2 cross-sections. The HRL for 1,3-dichloropropene is 0.4 µg/L.

Additional Monitoring in Conjunction with UCMR 1 Monitoring

UCMR 1 monitoring was conducted primarily from 2001 to 2003. Although 1,3-dichloropropene was not officially a UCMR 1 contaminant, EPA collected 1,3-dichloropropene data from UCMR 1 small system samples alongside the regular List 1 contaminants, using an appropriate analytical method that does not involve sodium sulfate or sodium thiosulfate. The surface water and ground water systems were selected to be representative of small systems nationwide. For a description of the UCMR 1 monitoring plan, see Chapter 2. See also USEPA (2006c) for more information on UCMR 1.

A total of 3,719 samples from 796 systems were analyzed for *cis*- and *trans*-1,3-dichloropropene. Neither isomer was detected in any sample. The reporting limit for each isomer was 0.50 µg/L. See Exhibit 6-12.

Exhibit 6-12: Summary UCMR 1 Occurrence Statistics for 1,3-Dichloropropene in Small Systems

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,719		--
Percent of Samples with Detections	0.00%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	0.4 µg/L		--
Minimum Reporting Level (MRL)	0.50 µg/L		--
99th Percentile Concentration of Detections	< MRL		--
Median Concentration of Detections	< MRL		--
Total Number of PWSs	796		60,414
Number of GW PWSs	589		56,072
Number of SW PWSs	207		4,342
Total Population	2,758,082		45,414,590
Population of GW PWSs	1,937,327		36,224,336
Population of SW PWSs	820,755		9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%	0
PWSs (GW & SW) > 1/2 HRL	0	0.00%	0
PWSs (GW & SW) > HRL	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	0	0.00%	0
Population Served by PWSs > 1/2 HRL	0	0.00%	0
Population Served by PWSs > HRL	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Pesticides in Ground Water Database (PGWDB)

The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992). Most of the data are from drinking water wells. Since PGWDB data come from multiple sources, they should be interpreted with caution. Results might be biased high, because areas with suspected contamination are likely to have been sampled more frequently than pristine areas. For more information on PGWDB, see Chapter 2.

According to the data compiled in the PGWDB, 1,3-dichloropropene was detected in 6 (0.03 percent) of 21,270 wells sampled. The detections were found in 3 out of 7 States where 1,3-dichloropropene was investigated. All three States with detections had concentrations higher than the HRL of 0.4 µg/L. Concentrations at three California wells ranged from 0.890 µg/L to 31.0 µg/L; concentrations at two Florida wells ranged from 0.279 µg/L to 7.83 µg/L; and concentrations at one New York well ranged from 18 to 140 µg/L (USEPA, 1992).

National Pesticide Survey (NPS)

EPA collected samples from approximately 1,300 CWS wells and rural drinking water wells between 1988 and 1990 for the National Pesticide Survey (NPS). The survey was designed to provide a statistically reliable estimate of pesticide occurrence in the nation's drinking water wells. For details about NPS, see Chapter 2.

Cis- and *trans*-1,3-dichloropropene were included in the survey as separate analytes, each with a minimum reporting limit of 0.010 µg/L. Neither compound was detected in the survey (USEPA, 1990).

Monitoring by Registrant

As a condition of re-registriation in 1998, Dow AgroSciences agreed to conduct tap water monitoring for 1,3-dichloropropene and its alcohol and acid degradates. High-use areas were to be targeted. It was decided that risk reduction measures would be implemented if levels exceeded 0.2 µg/L (USEPA, 1998).

Monitoring was conducted between April 2000 and April 2001 in five regions: the Central Columbia Plateau, Upper Snake River Basin, North Platter River, Albermarle-Pamlico Sound, and the Georgia/Florida basins. Approximately 5,800 samples were taken from 518 wells considered vulnerable to 1,3-dichloropropene contamination. Samples were tested for 1,3-dichloropropene, and two metabolites, 3-chloroallyl alcohol (CAAL) and 3-chloroacrylic acid (CAAC). Limits of detection (LODs) for the parent, CAAL, and CAAC were 0.015 µg/L, 0.023 µg/L, and 0.023 µg/L, respectively, and limits of quantitation (LOQs) were 0.05 µg/L, 0.092 µg/L, and 0.046 µg/L, respectively. Each well was sampled approximately four times (USEPA, 2004).

Of approximately 5,800 samples, 68 had at least one of the compounds in detectable quantities. These detections came from 65 of the 518 wells. Three wells had more than one

detection, but no well had more than two. There were 4 detections of 1,3-dichloropropene, with a maximum concentration of 0.145 µg/L; 14 detections of CAAL, with a maximum concentration of 0.11 µg/L; and 50 detections of CAAC, with a maximum detection of 0.12 µg/L. All detected concentrations were less than 0.2 µg/L, so no further action was required of the registrant (USEPA, 2004).

6.4 Technology Assessment

6.4.1 Analytical Methods

Analytical methods for 1,3-dichloropropene are readily available. EPA Methods 502.2 and 524.2 rely on purge and trap gas chromatography (GC), with detection accomplished using an electrolytic conductivity detector (ELCD) or a mass spectrometer (MS), respectively. Description of these methods can be found in EPA's *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III,* available from the Drinking Water Public Docket or the National Technical Information Service (NTIS), NTIS PB91-231480 (USEPA, 1995a). Historically, Methods 502.1 and 524.1 were also used to collect occurrence data for 1,3-dichloropropene. These methods were based on similar technology to Methods 502.2 and 524.2, but their approval for use in compliance monitoring of VOCs was withdrawn as of July 1, 1996.

The method detection limit (MDL) and the average recovery for each analytical method that can be used for the analysis of 1,3-dichloropropene in water are included in the method descriptions below[1].

Current versions of Methods 502.2 and 524.2 use either sodium thiosulfate or ascorbic acid for reducing free chlorine at the time of sample collection. However, there is evidence that 1,3-dichloropropene is unstable in the presence of sodium thiosulfate (Vuong *et al.*, 1998). While the current version of Method 524.2 does specify that only the ascorbic acid option should be used if samples are being collected for 1,3-dichloropropene analysis, previous versions of 524.2 did not include that requirement. Both the current and previous versions of Method 502.2 also do not include that requirement. Therefore, any sample that used sodium thiosulfate (or sodium sulfate) as a dechlorinating agent may yield an analytical result which underestimates the concentration of 1,3-dichloropene present in the sample.

[1] The Method Detection Limit (MDL) is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon a series of replicate measurements of the analyte at low concentrations. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

The average recovery is the fraction or percent concentration of a target analyte determined relative to the true or expected concentration from a sample containing a known amount of the target analyte. (This can result in apparent recovery values greater than 100 percent.)

EPA Method 502.2

EPA Method 502.2 (Revision 2.1), entitled "*Volatile Organic Compounds in Water by Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series,*" determines the presence of VOCs in water samples using GC in conjunction with either an ELCD or a photoionization detector (PID). Either detector may be used to detect and quantify *cis*- and *trans*-1,3-dichloropropene with similar sensitivity.

The MDL for *cis*- and *trans*-1,3-dichloropropene Method 502.2 is reported to range from 0.06 to 0.10 µg/L depending on the method option used. The average recovery for *cis*- and *trans*-1,3-dichloropropene using Method 502.2 is reported to range from 97 to 99 percent, depending on the method option (USEPA, 1995b).

EPA Method 524.2

EPA Method 524.2 (Revision 4.1), entitled "*Volatile Organic Compounds in Water by Purge and Trap Capillary Column Gas Chromatography/Mass Spectrometry*," is used to detect volatile aromatic compounds in finished drinking water, raw source water, or drinking water in any treatment stage. VOCs such as DCP are extracted by bubbling an inert gas through the aqueous sample. Purged sample components are trapped in a tube containing suitable sorbent materials. When purging is complete, the sorbent tube is heated and backflushed with helium to thermally desorb trapped sample components onto a capillary GC column. The column is temperature-programmed to separate the method analytes, which are then detected with a mass spectrometer. Analytes are identified and quantitated by comparison to standard materials (USEPA, 1995c).

MDLs for *cis*- and *trans*-1,3-dichloropropene are reported as 0.02 and 0.048 µg/L, respectively. The average recovery values are reported as 100 and 110 percent, respectively (USEPA, 1995c).

6.4.2 Treatment Technologies

Treatment technology status does not influence the determination of whether or not a contaminant should be regulated. However, treatment technologies must be readily available before a contaminant can be regulated with an NPDWR. There is no evidence that 1,3-dichloropropene is substantially removed by conventional treatments, such as coagulation/flocculation, sedimentation, and inert media filtration. Potential treatment technologies include air stripping and activated carbon.

Air stripping involves the continuous contact of air with the water being treated, allowing dissolved volatile contaminants to transfer from the source water to the air. Systems often consist of a large column (or tower) filled with molded plastic or ceramic packing material. As the water flows along the column, air is forced counter-current through the water. The packing material increases the area of air-liquid interface, enhancing mass transfer. After contact, the air is vented to an additional treatment device that safely contains or destroys the contaminant.

The Henry's Law constant is commonly used to indicate the tendency of a contaminant to partition from water to air. A larger Henry's constant indicates a greater equilibrium concentration of the contaminant in the air. Thus, contaminants with larger Henry's constants are more efficiently removed by air stripping. A compound is generally considered amenable to air stripping if it has a Henry's constant above that of dibromochloropropane (0.003 mol/mol) or ethylene dibromide (0.013 mol/mol) (Speth *et al.*, 2001). Speth *et al.* (2001) compiled Henry's Law constants, both calculated by the authors and reported in the literature, for Contaminant Candidate List (CCL) compounds. According to Speth *et al.* (2001), the Henry's Law constant for 1,3-dichloropropene is 0.088 mol/mol or 0.14 mol/mol, both of which indicate that air stripping is a viable treatment option.

Granular activated carbon (GAC) treatment removes contaminants via the physical and chemical process of sorption: the contaminants attach to the carbon surface as water passes through the carbon bed. Activated carbon has a large sorption capacity for many water impurities, including synthetic organic chemicals, taste- and odor-causing compounds, and some species of mercury.

Adsorption capacity is typically represented by the Freundlich isotherm constant, with higher Freundlich (K) values indicating greater sorption potential. Activated carbon is considered to be cost-effective for removing a particular contaminant if the Freundlich (K) value of the contaminant is above 200 $\mu g/g\ (L/\mu g)^{1/n}$ (Speth *et al.*, 2001). Gardner *et al.* (1990 as cited in Speth *et al.*, 2001) report that the Freundlich (K) value for 1,3-dichloropropene is 200 $\mu g/g\ (L/\mu g)^{1/n}$, which indicates that GAC might be a viable treatment option.

6.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate 1,3-DCP with a national primary drinking water regulation (NPDWR). Because 1,3-DCP appears to occur infrequently at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. While 1,3-DCP was detected in the UCM Round 1 (late 1980's) and the UCM Round 2 (mid 1990's) surveys, it was not detected in a subsequent evaluation of 796 small systems from the UCMR 1 survey. In addition, the USGS did not detect 1,3-DCP in two occurrence studies performed between 1999 and 2001 using monitoring levels that were lower than the HRL. EPA believes the 1999 pesticide labeling requirements, which are intended to mitigate risks to drinking water, may be one reason for the lack of occurrence of 1,3-DCP at levels of concern in subsequent monitoring surveys.

EPA recognizes that 1,3-dichloropropene is listed as a probable human carcinogen. For this reason, the Agency encourages those States with public water systems that may have 1,3-dichloropropene above the HRL to evaluate site-specific protective measures and to consider whether State-level (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory document for 1,3-DCP to provide more recent health information. The updated Health Advisory will provide information to any States with public water systems that may have 1,3-DCP above the HRL.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

6.6 References

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. *Toxicological Profile for 1,3-dichloropropene*. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services. Available on the Internet at: http://www.atsdr.cdc.gov/toxprofiles/tp40.html.

Albrecht, W.N. 1987. *Archives of Environmental Health*. Vol. 41, pp. 292-296. (As cited in HSDB, 2004).

Ashford, R.D. 1994. *Ashford's Dictionary of Industrial Chemicals*. London, England: Wavelength Publications, Ltd. (As cited in HSDB, 2004).

Baldys, S, III, T.H. Raines, B.L. Mansfield, and J.T. Sandlin. 1998. *Urban stormwater quality, event mean concentrations, and estimates of stormwater pollutant loads, Dallas-Fort Worth area, Texas, 1992–93.* U.S. Geological Survey. WRIR 98-4158, 51 p.

Batzer, F. R., J. L. Belcer, J. R. Peterson, and J. D. Wolt. 1997. Degradation of 1,3-dichloropropene in aerobic soils, Ch. 7, pp. 60-78 *In* J. N. Seiber, J. E. Woodrow, M. V. Yates, J. A. Knuteson, and S. R. Yates (eds.) *Fumigants: Environmental Fate, Exposure, and Analysis*. ACS Symposium Series 652, American Chemical Society. pp. 60-78. (As cited in HSDB, 2004).

Budavari, S. (ed.). 1996. *The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals*. Whitehouse Station, NJ: Merck & Co., Inc. (As cited in HSDB, 2004).

Cole, R.H., R.E. Frederick, R.P. Healy, and R.G. Rolan. 1984. Preliminary findings of the priority pollutant monitoring project of the Nationwide Urban Runoff Program. *Journal of the Water Pollution Control Federation*, 56(7):898-908.

Delzer, G.C. and T. Ivahnenko. 2003. *Occurrence and Temporal Variability of Methyl tert-Butyl Ether (MTBE) and Other Volatile Organic Compounds in Select Sources of Drinking Water: Results of the Focused Survey.* U.S. Geological Survey Water-Resources Investigations Report 02-4084, 65 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4084.pdf.

Gardner, D.A., G.M. Shaul, and K.A. Dostal. 1990. "Activated Carbon Isotherms for Pesticides." USEPA Final Report, Contract #68-03-3371, Risk Reduction Engineering Laboratory, Cincinnati, OH. (As cited in Speth *et al.*, 2001).

Gerhartz, W. (exec. ed.) 1985. *Ullmann's Encyclopedia of Industrial Chemistry*. 5th Edition, Vol. A1: Deerfield Beach, FL. VCH Publishers, 1985 to present. (As cited in HSDB, 2004).

Grady, S.J. 2003. *A National Survey of Methyl tert-Butyl Ether and Other Volatile Organic Compounds in Drinking-Water Sources: Results of the Random Survey.* U.S. Geological Survey

Water-Resources Investigations Report 02-4079, 85 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4079.pdf.

Hazardous Substances Data Bank (HSDB). 2004. Search for 1,3-dichloropropene. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed October 25, 2004.

Ivahnenko, T., S.J. Grady, and G.C. Delzer. 2001. *Design of a National Survey of Methyl tert-Butyl Ether and Other Volatile Organic Compounds in Drinking-Water Sources.* U.S. Geological Survey Open-File Report 01-271, 42 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr01_271.pdf.

Krijgsheld, K.R. and A. van der Gen. 1986. Assessment of the impact of the emission of certain organochlorine compounds on the aquatic environment: Part II: Allylchloride, 1,3- and 2,3-dichloropropene. *Chemosphere*. 15(7):861-880. (As cited in HSDB, 2004).

Leistra, M. 1970. Distribution of 1,3-dichloropropene over the phases in soil. *Journal of Agricultural and Food Chem*istry. 18:1124 - 1126. (As cited in ATSDR, 1992).

Lewis, R.J., Sr., ed. 1997. *Hawley's Condensed Chemical Dictionary.* 13th Ed. New, York, NY: John Wiley & Sons, Inc. (As cited in HSDB, 2004).

Lomax, L.G., W.T. Stott, K.A. Johnson, L. L. Calhoun, B. L. Yano, and J. F. Quast. 1989. The chronic toxicity and oncogenicity of inhaled technical grade 1,3-dichloropropene in rats and mice. *Fundamental and Applied Toxicology.* 12:418-431. (As cited in USEPA, 2000).

Lopes, T.J., K.D. Fossum, J.V. Phillips, and J.E. Monical. 1995. *Statistical summary of selected physical, chemical, and microbial characteristics, and estimates of constituent loads in urban stormwater, Maricopa County, Arizona.* U.S. Geological Survey Water-Resources Investigations Report 94-4240.

Lopes, T.J. and S.G. Dionne. 1998. *A Review of Semivolatile and Volatile Organic Compounds in Highway Runoff and Urban Stormwater.* U.S. Geological Survey Open-File Report 98-409, 67 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr98-409.pdf.

Lyman, W.J, W.F. Reehl and DH. Rosenblatt. 1990. *Handbook of Chemical Property Estimation Methods.* Washington, DC: American Chemical Society, pp. 4-9, 15-1 to 15-29. (As cited in HSDB, 2004).

Munnecke, D.E. and S.D. Vangundy. 1979. Movement of fumigants in soil, dosage, responses, and differential effects. *Annual Review of Phytopathology*. 17:405-429. (As cited in ATSDR, 1992).

National Center for Food and Agricultural Policy (NCFAP). 2003. National Pesticide Use Database. Available on the Internet at: http://www.ncfap.org/database/default.htm.

National Institute of Occupational Safety & Health (NIOSH). 2004. *Pocket Guide to Chemical Hazards*. Washington, DC: U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health. DHHS (NIOSH) Publication No. 97-140. Available on the Internet at: http://www.cdc.gov/niosh/npg/npg.html.

National Toxicology Program (NTP). 1985. *Toxicology and carcinogenesis studies of Telone II (technical grade 1,3-dichloropropene containing 1% epichlorohydrin as a stabilizer) in F344/N rats and B6C3F1 mice (gavage studies).* (As cited in USEPA, 2000).

Roberts, T.R. and G. Stoydin. 1976. The degradation of (Z)- and (E)-1,3- dichloropropenes and 1,2-dichloropropane in soil. *Pesticide Science*. 7:325-335. (As cited in HSDB, 2004).

Sittig, M. (ed.). 1980. *Pesticide Manufacturing and Toxic Materials Control Encyclopedia*. Park Ridge, NJ: Noyes Data Corporation. (As cited in HSDB, 2004).

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

Squillace, P.J., M.J. Moran, W.W. Lapham, C.V. Price, R.M. Clawges, and J.S. Zogorski. 1999. Volatile organic compounds in untreated ambient groundwater of the United States, 1985-1995. *Environmental Science and Technology*. 33(23):4176-4187. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/journal/EST.voc.squillace.pdf.

Stott, W.T., K.A. Johnson, T.K. Jeffries, K.T. Haut, and S.N. Shabrang. 1995. *Telone II soil fumigant: two-year chronic toxicity/oncogenicity study in Fischer 344 rats*. Study # M-003993-031l. (As cited in USEPA, 2000).

Swann, R.L., D.A. Laskowski, P.J. McCall, K. Vander Kuy, and H.J. Dishburger. 1983. A rapid method for the estimation of the environmental parameters octanol/water partition coefficient, soil sorption constant, water to air ratio and water solubility. *Residue Reviews*. 85(12):17-28. (As cited in ATSDR, 1992).

Thelin, G.P. and L.P. Gianessi. 2000. *Method for Estimating Pesticide Use for County Areas of the Conterminous United States.* U.S. Geological Survey Open-File Report 00-250, 62 p. Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/ofr00250/ofr00250.pdf.

Tomlin, C.D.S. 1997. *The Pesticide Manual - World Compendium*, 11th Edition, British Crop Protection Council, Surrey, England. (As cited in HSDB, 2004).

United States Environmental Protection Agency (USEPA). 1980. *Ambient Water Quality Criteria Document: Dichloropropanes/Dichloropropenes (Draft)*. (As cited in HSDB, 2004).

USEPA. 1990. *National Pesticide Survey: Survey Analytes.* EPA Report 570-9-90-NPS2. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS2.]

USEPA. 1992. *Pesticides in Ground Water Database: A Compilation of Monitoring Studies, 1971-1991. National Summary.* Office of Prevention, Pesticides and Toxic Substances. EPA Report 734-12-92-001. Available on the Internet at: http://nepis.epa.gov/pubtitleOPPTS.htm. [Search for document number 7341292001.]

USEPA. 1995a. *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III*. EPA Report 600-R-95-131. August, 1995.

USEPA. 1995b. Method 502.2 - Volatile Organic Compounds in Water by Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series. Revision 2.1. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III*. EPA Report 600-R-95-131. August, 1995.

USEPA. 1995c. Method 524.2 - Measurement of Purgeable Organic Compounds in Water by Capillary Column Gas Chromatography/Mass Spectrometry. Revision 4.1. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III*. EPA Report 600-R-95-131. August, 1995.

USEPA. 1998. *Reregistration Eligibility Decision (RED)--1,3-Dichloropropene*. EPA Report 738-R-98-016. Washington, DC: Office of Prevention, Pesticides and Toxic Substances. December 1998. Available on the Internet at: http://www.epa.gov/oppsrrd1/REDs/0328red.pdf.

USEPA. 2000. Integrated Risk Information Systems (IRIS), 1,3-Dichloropropene. May 2000. Available on the Internet at: http://www.epa.gov/iris/subst/0224.htm. Accessed February 2, 2005.

USEPA. 2004e. *Memorandum - EFED Review of a Telone Well Monitoring Study.* Office of Prevention, Pesticide, and Toxic Substances. March 10, 2004.

USEPA. 2006a. TRI Explorer: Trends. Search for 1,3-dichloropropylene. Available on the Internet at: http://www.epa.gov/triexplorer/trends.htm. Accessed February 8, 2006. Last modified June 8, 2005.

USEPA. 2006b. *The Analysis of Occurrence Data from the Unregulated Contaminant Monitoring (UCM) Program and National Inorganics and Radionuclides Survey (NIRS) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-009. Draft. December 2006.

USEPA. 2006c. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

United States Geological Survey (USGS). 2004. 1997 Pesticide Use Maps. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pesticide_use_maps_1997/. Accessed June 3, 2004.

van der Pas, L.J.T. and M. Leistra. 1987. Movement and transformation of 1,3-dichloropropene in the soil of flower-bulb fields. *Archives of Environmental Contamination and Toxicology*. 16:417-422. (As cited in HSDB, 2004).

von Guerard, P. and W.B. Weiss. 1995. *Water quality of storm runoff and comparison of procedures for estimating storm-runoff loads, volume, event-mean concentrations, and the mean load for a storm for selected properties and constituents for Colorado Springs, southeastern Colorado, 1992*. U.S. Geological Survey Water-Resources Investigations Report 94-4194.

Vuong, L. *et al.* 1998. EPA 524.2 Method: Comparison Study on Current Preservation Reagents Versus Sodium Bisulfate. Proceedings of the American Water Works Association Water Quality Technology Conference, November 1-4, San Diego, CA.

Warner, M. L.P., J.M. Cohon, and J.C. Ireland. 1987. *Determination of Henry's Law Constants of Selected Priority Pollutants*. USEPA/600/D-87/229, Washington, DC. NTIS PB87-212684. (As cited in HSDB, 2004).

Yon, D.A., G.A. Morrison, and A.S. McGibbon. 1991. The dissipation of 1,3-dichloropropene in ditch bottom sediment and associated aerobic ditch water. *Pesticide Science*. 32(147-159). (As cited in HSDB, 2004).

Chapter 7:
2,4- and 2,6-Dinitrotoluene

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

2,4- and 2,6-Dinitrotoluene (DNT), semi-volatile organic compounds (SVOCs), are two of the most common of the six isomers of dinitrotoluene. Dinitrotoluenes are used in the production of polyurethane foams, automobile air bags, dyes, ammunition, and explosives, including trinitrotoluene (TNT). Neither 2,4- nor 2,6-DNT occurs naturally. They are generally produced as individual isomers or as a mixture called technical grade DNT (tg-DNT). Technical grade DNT contains approximately 76 percent 2,4-DNT and 19 percent 2,6-DNT, with the remainder consisting of the other isomers and minor contaminants.

In chronic exposures, oral dietary administration of 2,4-DNT to dogs primarily affected the nervous system, erythrocytes, and biliary tract. A study in dogs found a lowest-observed-adverse-effect level (LOAEL) of 1.5 mg/kg/day and a no-observed-adverse-effect level (NOAEL) of 0.2 mg/kg/day. Observed effects included neurotoxicity, hematologic changes, and effects on bile ducts. EPA established a reference dose (RfD) of 0.002 mg/kg/day for 2,4-DNT based on this study. An uncertainty factor of 100, to account for interspecies and intraspecies variability, was applied to derive the RfD.

EPA established an RfD of 0.001 mg/kg/day for 2,6-DNT. This RfD was based on neurotoxicity, Heinz body formation, biliary tract hyperplasia, liver and kidney histopathology, and death in beagle dogs that were fed gelatin capsules containing 2,6-DNT daily for up to 13 weeks. The NOAEL for this study was 4 mg/kg/day, and an uncertainty factor of 3,000 (100 for inter- and intra-species variability, 10 for the use of a subchronic study, 3 to account for the limited database) was applied to derive the RfD.

DNT is considered likely to be carcinogenic to humans (it is classified as a B2 carcinogen). This determination is based on significant increases in hepatocellular carcinoma and mammary gland tumors in female rats fed a DNT mixture (98 percent 2,4-DNT with 2 percent 2,6-DNT) in the diet in a two-year study. Concentrations of 5 µg/L, 0.5 µg/L, and 0.05 µg/L are associated with carcinogenic risks of 10^{-4}, 10^{-5}, and 10^{-6} respectively.

2,4-DNT has been shown to cause reproductive effects in rats, mice, and dogs. There are currently no studies on the reproductive or developmental toxicity of 2,6-DNT. A study of tg-DNT administered to rats in corn oil by gavage found significant increases in relative liver and spleen weight in the fetuses of dams administered DNT at levels of 35 mg/kg/day or greater. No teratogenic toxicity was seen in the study rats.

DNT toxicity might be different in children, compared to adults, since it undergoes bioactivation in the liver and by the intestinal microflora. Newborns might be more sensitive to DNT-related methemoglobinemia because an enzyme that protects against increased levels of methemoglobin is inactive for a short duration immediately after birth. However, there are no empirical data on differences in children's responses to 2,4-/2,6-DNT.

No recent quantitative estimates of DNT production or use are available. According to one older estimate, combined 2,4- and 2,6-DNT production amounted to 272,610,000 pounds in 1975. Estimates of industrial releases of 2,4-DNT and 2,6-DNT are available from 1988 to 2003

through the Toxic Release Inventory (TRI). Releases of both chemicals declined in the early 1990s, and then peaked again around 1999-2001. On-site air emissions and surface water releases were generally the most consistently reported types of releases, with surface water releases generally declining over the period on record. In addition, TRI lists mixed DNT isomer releases as a separate category over the same time period. Underground injections made up the bulk of on-site releases during the 1990s, but diminished thereafter. Total releases peaked in 1993 and 1997, and generally diminished in recent years.

The United States Geological Survey (USGS) has collected data on the ambient occurrence of these contaminants. A study of bed sediments from representative watersheds across the country found 2,6-DNT in between 1.6% and 6.9% of samples collected in various land-use settings. In all land-use settings, most detected concentrations of 2,6-DNT were below the reporting limit. Detections of 2,4-DNT in bed sediment were much less frequent. In addition, a USGS review of highway and urban runoff studies shows no detects of either 2,4- or 2,6-DNT.

To determine the extent of 2,4- and 2,6-DNT contamination in drinking water, EPA included these contaminants as analytes in the first Unregulated Contaminant Monitoring Regulation (UCMR 1). Because the health reference level (HRL) for both 2,4- and 2,6-DNT (0.05 µg/L) is lower than the minimum reporting level (MRL) of 2 µg/L used for monitoring, EPA used the MRL to evaluate occurrence and exposure. The MRL is within the 10^{-4} to the 10^{-6} cancer risk range for 2,4- and 2,6-DNT. In evaluating the UCMR 1 data, EPA found that 1 of the 3,866 public water systems (PWSs) sampled (or 0.03 percent) detected 2,4-DNT at or above the MRL of 2 µg/L, affecting 0.02 percent of the population served (or 38,000 people from 226 million). None of the 3,866 PWSs sampled (serving 226 million) detected 2,6-DNT at or above the MRL of 2 µg/L.

The Agency has made a preliminary determination not to regulate 2,4- or 2,6-DNT with a national primary drinking water regulation (NPDWR). Because 2,4- and 2,6-DNT appear to occur infrequently at levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

EPA recognizes that 2,4- and 2,6-DNT are listed as probable human carcinogens. For this reason, the Agency encourages those States with public water systems that may have either 2,4- or 2,6-DNT above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency's original Health Advisories for 2,4 and 2,6-DNT were developed for military installations. Because the Agency recognizes that 2,4 and 2,6-DNT may still be found at some military sites, the Agency has updated the Health Advisories to reflect recent health effects publications. The Health Advisories are available for review in the docket. The updated Health Advisories will provide information to any States with public water systems that may have either 2,4- or 2,6-DNT at concentrations above the HRL.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

AOAC	Association of Official Analytical Chemists
APHA	American Public Health Association
ASTM	American Society for Testing and Materials
CAS	Chemical Abstracts Service
CCL	Contaminant Candidate List
CWS	Community Water System
DNT	Dinitrotoluene
2,4-DNT	2,4-Dinitrotoluene
2,6-DNT	2,6-Dinitrotoluene
GAC	Granular Activated Carbon
GC	Gas Chromatography
GC/MS	Gas Chromatography with Mass Spectrometry
HRL	Health Reference Level
HSDB	Hazardous Substances Data Bank
LOAEL	Lowest Observed Adverse Effect Level
LSE	Liquid-Solid Extraction
MDL	Method Detection Limit
MRL	Minimum Reporting Level
MS	Mass Spectrometry
NAWQA	National Water Quality Assessment
NOAEL	No Observed Adverse Effect Level
NPDES	National Pollutant Discharge Elimination System
NPDWR	National Primary Drinking Water Regulation
NPL	National Priorities List
NTNCWS	Non-Transient Non-Community Water System
PWS	Public Water System
QC	Quality Control
RCRA	Resource Conservation and Recovery Act
RfD	Reference Dose
RL	Reporting Limit
SVOC	Semi-Volatile Organic Compound
tg-DNT	Technical Grade DNT
TNT	Trinitrotoluene
TRI	Toxics Release Inventory
UCMR 1	First Unregulated Contaminant Monitoring Regulation
USGS	United States Geological Survey

7 2,4- and 2,6-Dinitrotoluene

7.1 Definition

2,4- and 2,6-Dinitrotoluene are semivolatile organic compounds (SVOCs) with very similar physical characteristics. 2,4- and 2,6-Dinitrotoluene are just two of the six isomers of dinitrotoluene (DNT), but together they comprise approximately 95 percent of technical grade dinitrotoluene (ATSDR, 1998). The remaining 5 percent is composed primarily of the other four isomers (2,3-dinitrotoluene, 2,5-dinitrotoluene, 3,4-dinitrotoluene, and 3,5-dinitrotoluene). 2,4-Dinitrotoluene's Chemical Abstracts Service (CAS) registry number is 121-14-2, and 2,6-Dinitrotoluene's number is 606-20-2. There are multiple synonyms for 2,4-dinitrotoluene: 2,4-DNT, 1-methyl-2,4-dinitrobenzene, 2,4-dinitrotoluol, NCI-C01865, dinitrotoluene, and Resource Conservation and Recovery Act (RCRA) waste number U105. 2,6-Dinitrotoluene is also known as: 2,6-DNT, 1-methyl-2,6-dinitrobenzene, RCRA waste number U106, and 2-methyl-1,3-dinitro-benzene (NIST, 2001).

7.1.1 Properties and Sources

In pure form, both 2,4- and 2,6-dinitrotoluene are pale yellow solids with a slight odor. 2,4- and 2,6-Dinitrotoluene are not natural substances, but are made from reacting toluene (C_7H_8) with a mixture of nitric and sulfuric acids. 2,4- and 2,6-Dinitrotoluene are commonly used in the bedding and furniture industries to produce polyurethane foams; however, they are also used in the production of ammunition, explosives, dyes, and can be found in automobile air bags (ATSDR, 1998). The two contaminants are released to the environment predominantly through industrial wastewater discharges and improper waste disposal. Exhibit 7-1 summarizes the physical and chemical properties of 2,4- and 2,6-dinitrotoluene.

Exhibit 7-1: Physical and Chemical Properties of 2,4- and 2,6-Dinitrotoluene

Identification	2,4-Dinitrotoluene	2,6-Dinitrotoluene
CAS number	121-14-2	606-20-2
Molecular Formula	$C_7H_6N_2O_4$	$C_7H_6N_2O_4$
Physical and Chemical Properties		
Boiling Point	300 °C [1]	285 °C [9]
Melting Point	71 ° C [1]	66 °C [1]
Molecular Weight	182.14 g/mol [1]	182.14 g/mol [1]
Log K_{oc}	2.45 [2]	2.31 [2]
Log K_{ow}	1.98 [3]	2.10 [10]
Water Solubility	270 mg/L at 22 °C [4]	180 mg/L at 20° C [11]
Vapor Pressure	1.4×10^{-4} mm Hg at 22 ° C [5]	5.67×10^{-4} mm Hg at 25 ° C [5]
Henry's Law Constant	8.67×10^{-7} atm-m^3/mol [6] 4.6×10^{-5} (dimensionless), predicted [7]	2.17×10^{-7} atm-m^3/mol [12] 0.22 (dimensionless), predicted [7] 7.4 (dimensionless), from literature [7]
Freundlich Isotherm Constant (K)	17,200 $(\mu g/g)(L/\mu g)^{1/n}$ [8]	15,900 $(\mu g/g)(L/\mu g)^{1/n}$ [8]

[1] *Lide, 1999 (as cited in HSDB, 2004)*

[2] *Lyman, 1982 (as cited in Howard, 1990)*

[3] *Hansch et al., 1995 (as cited in HSDB, 2004)*

[4] *Spanggord et al., 1980 (as cited in HSDB, 2004)*

[5] *Pella, 1977 (as cited in HSDB, 2004)*

[6] *Smith et al., 1983 (as cited in Howard, 1990)*

[7] *Speth et al., 2001*

[8] *Dobbs and Cohen, 1980 (as cited in Speth et al., 2001)*

[9] *USEPA, 1980 (as cited in HSDB, 2004)*

[10] *Nakagawa et al., 1992 (as cited in HSDB, 2004)*

[11] *Mabey et al., 1982 (as cited in ATSDR, 1998)*

[12] *SGC, 1987 (as cited in Howard, 1990)*

7.1.2 Environmental Fate and Behavior

Both 2,4- and 2,6-dinitrotoluene are slightly mobile in soil (Howard, 1990). Degradation in soil is fairly rapid, as both compounds are broken down by sunlight and bacteria into

substances such as carbon dioxide, water, and nitric acid (ATSDR, 1998). At a munitions-contaminated site, microorganisms in the surface soil were reported to transform 2,4- and 2,6-dinitrotoluene to amino-nitro intermediates within 70 days (Bradley *et al.*, 1994 as cited in ATSDR, 1998). This process could take longer or shorter, however, as natural degradation of both compounds in soil has been found to be temperature-sensitive (Grant *et al.*, 1995 as cited in ATSDR, 1998). Aromatic nitro compounds such as 2,4- and 2,6-dinitrotoluene are not susceptible to hydrolysis (Lyman *et al.*, 1982 as cited in Howard, 1990).

In water, both 2,4- and 2,6-dinitrotoluene have a slight tendency to adsorb to sediments and suspended solids (Howard, 1990). Volatilization from water does not appear to be a significant transport process for either contaminant (Howard, 1990). Available data on 2,4- and 2,6-dinitrotoluene degradation in water are variable and inconsistent (Howard, 1990). Jenkins *et al.* (1995) note that rates of biodegradation of nitrotoluenes and similar compounds are sufficient to require that special steps be taken to preserve aqueous samples for laboratory analysis. However, 2,4- and 2,6-dinitrotoluene have relatively long half-lives in aquatic systems, facilitating aquatic transport (ATSDR, 1998). Degradation of dinitrotoluene in water can occur via several mechanisms, including photolysis, microbial biodegradation, ozonation and chlorination, and oxidation by strong oxidants such as hydrogen peroxide, ozone, or oxone (ATSDR, 1998). Analyses of both contaminants' log K_{ow} suggest that the bioaccumulation potentials of 2,4- and 2,6-dinitrotoluene in aquatic organisms are quite low (Hansch *et al.*, 1995 as cited in HSDB, 2004).

According to a model of gas/particle partitioning for SVOCs (Bidleman, 1988 as cited in HSDB, 2004), dinitrotoluenes are expected to exist solely as vapor in the ambient atmosphere. Vapor-phase 2,4- and 2,6-dinitrotoluene are degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals. The half-life for this reaction is estimated to be 75 days (HSDB, 2004).

7.2 Health Effects

In experimental animal studies, 2,4- and 2,6-DNT appear to be acutely toxic at moderate to high levels (LD_{50}s[1] ranging from 180 to 1,954 mg/kg) when administered orally. In subacute studies (4 weeks) conducted by Lee *et al.* (1978 as cited in ATSDR, 1998), dogs, rats, and mice were fed 2,4-DNT and studied for toxic effects. A "no-observed-adverse-effect level" (NOAEL) of 5 mg/kg/day was established; decreased body weight gain and food consumption, neurotoxic signs, and lesions in the brain, kidneys, and testes occurred at 25 mg/kg/day (the highest dose tested).

Subchronic studies in mice, rats, and dogs that administered 2,4- and 2,6-DNT in the diet produced similar effects in all species. All species exposed to 2,4-DNT exhibited methemoglobinemia, anemia, bile duct hyperplasia sometimes accompanied by hepatic degeneration, and depressed spermatogenesis. Neurotoxicity and renal degeneration occurred in dogs at a dose level of 20 mg/kg/day of 2,6-DNT (Lee *et al.*, 1976 as cited in USEPA, 1992). At a dose level of 25 mg/kg/day of 2,4-DNT, male and female dogs developed impaired muscle

[1] LD_{50} = An estimate of a single dose that is expected to cause the death of 50 percent of the exposed animals. It is derived from experimental data.

movement and paralysis, methemoglobinemia, aspermatogenesis, hemosiderosis of the spleen and liver, cloudy swelling of the kidneys, and lesions of the brain (Ellis *et al.*, 1985 as cited in USEPA, 1992). These doses were determined to be "lowest-observed-adverse-effect levels" (LOAELs) for these studies.

2,4-DNT has been shown to cause reproductive effects in rats, mice, and dogs (Ellis *et al.*, 1979 as cited in USEPA, 1992; Lee *et al.*, 1985 as cited in ATSDR, 1998; Hong *et al.*, 1985 as cited in ATSDR, 1998; Ellis *et al.*, 1985 as cited in USEPA, 1992). Ellis *et al.* (1979 as cited in USEPA, 1992) observed effects in rats following dietary exposure after a dose of 35 mg/kg/day but not 5 mg/kg/day over three generations. Male mice fed 2,4-DNT for 13 weeks exhibited testicular degeneration and atrophy and decreased spermatogenesis at 95 mg/kg/day (Hong *et al.*, 1985 as cited in ATSDR). In another reproductive study, dogs exhibited mild to severe testicular degeneration and reduced spermatogenesis (Ellis *et al.*, 1985, as cited in USEPA, 1992) when administered 2,4-DNT in capsules at 25 mg/kg/day. There are currently no studies of the reproductive or developmental toxicity of 2,6-DNT, although a subchronic study in dogs identified atrophy of spermatogenic cells in males suggesting a one- or two-generation study as a data need for 2,6-DNT.

Some studies evaluated the effects of DNT in the form of a technical grade mixture (tg-DNT). In a study by Price *et al.* (1985 as cited in USEPA, 1992), the teratogenic potential of tg-DNT (containing approximately 76 percent 2,4-DNT and 19 percent 2,6-DNT) was investigated in rats. The study was conducted in two phases to evaluate the possible teratogenicity of DNT as well as DNT effects on postnatal development. For the first phase, rats were administered 0, 14, 35, 37.5, 75, 100, or 150 mg/kg/day of DNT in corn oil by gavage. In the postnatal phase, rats were administered 14, 35, 37.5, 75, or 100 mg/kg/day of DNT in corn oil by gavage. The NOAEL and LOAEL for developmental toxicity were 14 and 35 mg/kg/day, respectively, based on significant increases in relative liver and spleen weight in the fetuses of dams administered DNT at levels of 35 mg/kg/day or greater. No teratogenic toxicity was seen in the study rats.

In chronic exposures, oral dietary administration of 2,4-DNT to dogs primarily affected the nervous system, erythrocytes, and biliary tract (Ellis *et al.*, 1979; 1985 both as cited in USEPA, 1992). Based on neurotoxicity, hematologic changes, and effects on the bile ducts in dogs, the LOAEL was determined to be 1.5 mg/kg/day and the NOAEL was 0.2 mg/kg/day. EPA established a reference dose (RfD) of 0.002 mg/kg/day for 2,4-DNT (USEPA, 1992) based on this study. An uncertainty factor of 100, to account for interspecies and intraspecies variability, was applied to derive the RfD.

EPA established an RfD of 0.001 mg/kg/day for 2,6-DNT (USEPA, 1992). This RfD was also based on neurotoxicity, Heinz body formation, biliary tract hyperplasia, liver and kidney histopathology, and death in beagle dogs that were fed gelatin capsules containing 2,6-DNT daily for up to 13 weeks (Lee *et al.*, 1976 as cited in USEPA, 1992). The NOAEL for this study was 4 mg/kg/day, and an uncertainty factor of 3,000 (100 for inter- and intra-species variability, 10 for the use of a subchronic study, 3 to account for the limited database) was applied to derive the RfD.

DNT is likely to be carcinogenic to humans (classified as a B2 carcinogen; USEPA, 1990). This is based on significant increases in hepatocellular carcinoma and mammary gland

tumors in female rats fed DNT (98 percent 2,4-DNT with 2 percent 2,6-DNT) in the diet in a two-year study (Ellis *et al.*, 1979 as cited in USEPA, 1992). The tumor incidence in the female rats was used to establish a slope factor of 6.67×10^{-1} according to the 1999 EPA guidelines. Concentrations of 5 µg/L, 0.5 µg/L, and 0.05 µg/L are associated with carcinogenic risks of 10^{-4}, 10^{-5}, and 10^{-6} respectively. There were no studies found in the literature that evaluated the effects of 2,4- or 2,6-DNT on children. There is evidence that the pups and fetuses from dams administered tg-DNT had significant increases in relative liver and spleen weights (Price *et al.*, 1985 as cited in USEPA, 1992). DNT toxicity may be different in children, compared to adults, since it undergoes bioactivation in the liver and by the intestinal microflora (ATSDR, 1998). Newborns may be more sensitive to DNT-related methemoglobinemia because an enzyme that protects against increased levels of methemoglobin is inactive for a short duration immediately after birth (Gruener, 1976 as cited in ATSDR, 1998; ATSDR, 1998). However, there are no experimental data on differences in children's responses to 2,4-/2,6-DNT.

7.3 Occurrence and Exposure

7.3.1 Use and Environmental Release

Dinitrotoluenes (DNTs) are not known to occur naturally in the environment. Generally, 2,4- and 2,6-DNT are produced as a mixture called technical grade DNT, or simply DNT, which contains approximately 76 percent 2,4-DNT and 19 percent 2,6-DNT. The remainder of technical grade DNT consists of other isomers and minor contaminants such as trinitrotoluene (TNT) and mononitrotoluenes (HSDB, 2004). DNT is commercially produced by reacting toluene with a mixture of nitric and sulfuric acids (Etnier, 1987 as cited in ATSDR, 1998). DNT is used in the production of toluene diisocyanate and urethane polymers, as well as automobile airbags, dyes, and explosives, including TNT (ATSDR, 1998).

No recent quantitative estimates of DNT production or use are available. The Hazardous Substances Data Bank (HSDB, 2004) cites a 1980 EPA Ambient Water Quality Criteria Document that places combined 2,4- and 2,6-DNT production at 272,610,000 pounds in 1975.

2,4-DNT, 2,6-DNT, and mixed DNT are all listed as Toxics Release Inventory (TRI) chemicals. For a discussion of the nature and limitations of TRI data, see Chapter 2.

TRI data for 2,4-DNT (see Exhibit 7-2) are reported for the years 1988-2003. TRI releases for 2,4-DNT were reported from facilities in 21 States (AK, CA, FL, IA, IL, IN, KY, LA, MI, MO, MS, NE, NJ, NV, OH, SC, TN, TX, VA, UT, WV). Releases of all kinds declined in the early 1990s, and then peaked again around 1999-2001. On-site air emissions and surface water releases were generally the most consistent types of releases (USEPA, 2006a).

Exhibit 7-2: Environmental Releases (in pounds) of 2,4-Dinitrotoluene in the United States, 1988-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Total Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1988	93,257	12,055	106,400	14,961	124,281	350,954
1989	12,713	12,657	0	341	194,167	219,878
1990	57,593	3,735	74,000	2,153	99	137,580
1991	5,417	2,682	0	1,424	57	9,580
1992	1,764	105	0	0	0	1,869
1993	1,879	319	0	0	10	2,208
1994	1,899	399	0	0	255	2,553
1995	1,874	231	0	0	94	2,199
1996	1,891	349	0	0	0	2,240
1997	1,801	90	0	0	0	1,891
1998	1,995	187	0	10,000	1,408	13,590
1999	2,287	169	0	43,420	49,296	95,172
2000	1,931	177	250	27,609	19,601	49,568
2001	2,190	10	5	665,529	28,137	695,871
2002	205	6	0	0	2,381	2,592
2003	2,544	5	0	0	12,350	14,899

Source: USEPA, 2006a

TRI data for 2,6-DNT (see Exhibit 7-3) are also reported for the years 1988-2003. TRI releases for 2,6-DNT were reported from facilities in 10 States (AR, CA, IN, KY, LA, MI, NV, OH, TX, WV) with no more than nine States having reporting facilities in any one year. These data show a similar trend of declining releases in the late 1980s and early 1990s, and a subsequent peak around 2001. Again, on-site air emissions and surface water discharges are the most consistent types of release (USEPA, 2006a).

Exhibit 7-3: Environmental Releases (in pounds) of 2,6-Dinitrotoluene in the United States, 1988-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1988	87,597	957	27,000	0	30,882	146,436
1989	83,914	1,083	18,000	0	58,256	161,253
1990	17,737	416	19,000	0	0	37,153
1991	1,948	702	0	0	0	2,650
1992	425	126	0	0	0	551
1993	471	212	0	0	0	683
1994	516	374	0	0	0	890
1995	469	126	0	0	0	595
1996	472	94	0	0	0	566
1997	438	24	0	0	0	462
1998	472	62	0	0	0	534
1999	660	43	0	15,287	16,910	32,900
2000	513	32	250	0	2,030	2,825
2001	740	0	0	1,298,442	5,360	1,304,542
2002	117	1	0	0	855	973
2003	372	0	0	0	10,565	10,937

Source: USEPA, 2006a

TRI data for mixed dinitrotoluene isomers (see Exhibit 7-4) are reported for the years 1990-2003. TRI releases for mixed isomers were reported from facilities in 9 States (CA, IA, LA, NV, NJ, OH, OK, TX, UT) with no more than seven States having reporting facilities in any one year. Two States, Louisiana and Texas, reported releases every year. Underground injections made up the bulk of on-site releases during the 1990s, but diminished thereafter. Air emissions remained relatively constant. Surface water discharges and releases to land were generally insignificant but peaked in 2003. Off-site releases varied widely. Total releases peaked in 1993 and 1997, and generally diminished in recent years (USEPA, 2006a).

Exhibit 7-4: Environmental releases (in pounds) of Dinitrotoluene (Mixed Isomers) in the United States, 1990-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1990	4,159	7,112	0	363	15,832	27,466
1991	14,979	135	60,000	0	55	75,169
1992	16,744	291	50,000	0	61	67,096
1993	15,969	631	98,000	173	314	115,087
1994	15,930	10	28,000	0	6,515	50,455
1995	14811	284	17,000	0	6	32,101
1996	14,815	586	33,000	0	121	48,522
1997	11,551	63	56,000	0	46,491	114,105
1998	13,439	1	36,005	0	1,403	50,848
1999	9,657	1	1,100	0	322	11,080
2000	10,423	4	3,300	696	22,098	36,521
2001	9,839	8	3,000	15	696	13,558
2002	8,043	61	1,100	0	1,535	10,739
2003	6,767	1,318	190	4,110	1,405	13,790

Source: USEPA, 2006a.

2,4-DNT and 2,6-DNT have been detected in soil, sediment, water, or air at 69 and 53, respectively, of the 1,467 current or former National Priorities List (NPL) hazardous waste sites (HazDat, 1998 as cited in ATSDR, 1998).

7.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are sources of drinking water. Data on the occurrence of 2,4- and 2,6-dinitrotoluene in stream bed sediment are available from the National Water Quality Assessment (NAWQA) program of the United States Geological Survey (USGS). For details on this program, see the discussion in Chapter 2. Limited data on the occurrence of 2,4- and 2,6-DNT in ambient water are also available from stormwater studies.

NAWQA National Pesticide Synthesis: SVOCs in Bed Sediment

Because SVOCs like 2,4- and 2,6-dinitrotoluene are hydrophobic and tend to sorb to sediment and particles, an analysis of bed sediment is often the best way to determine whether an SVOC is present in water. The NAWQA National Pesticide Synthesis includes an analysis of SVOC monitoring in bed sediment from representative watersheds across the country between 1992 and 2001. Sampling was conducted at 1,029 sites. The reporting level for all SVOCs was 50 µg/L. Sampling techniques and analytical methods are described in detail by Nowell and Capel (2003).

NAWQA data indicate that 2,4-dinitrotoluene was not detected in bed sediment in agricultural, urban, or undeveloped settings (Exhibit 7-5). In mixed land use settings, 2,4-dinitrotoluene was detected in 1.3% of samples, with a maximum concentration of 173 µg/kg dry weight (Nowell and Capel, 2003).

Exhibit 7-5: USGS National Synthesis Summary of NAWQA Monitoring of 2,4-Dinitrotoluene in Bed Sediment, 1992-2001

Land Use Type	No. of Sites	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	242	0.0%	<RL	<RL	<RL
Urban	130	0.0%	<RL	<RL	<RL
Mixed	306	1.3%	<RL	<RL	173 µg/kg
Undeveloped	215	0.0%	<RL	<RL	<RL

Abbreviations:
RL = Reporting limit. Reporting limits for 2,4-dinitrotoluene varied, but did not exceed 50 µg/kg.

For bed sediment, all weights are dry weights.

Most sites were sampled only once. In the case of sites sampled multiple times, USGS used a single sample (the earliest sample with complete data for all analytes) to represent each site in this analysis.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Nowell and Capel, 2003

2,6-Dinitrotoluene was detected in bed sediment at frequencies ranging from 1.6% in urban settings to 4.4% in agricultural settings, 6.6% in mixed land use settings, and 6.9% in undeveloped settings (Exhibit 7-6). The 95th percentile concentrations were less than the reporting level in all settings. The highest concentration, 291 µg/kg dry weight, was found in an undeveloped setting (Nowell and Capel, 2003).

Exhibit 7-6: USGS National Synthesis Summary of NAWQA Monitoring of 2,6-Dinitrotoluene in Bed Sediment, 1992-2001

Land Use Type	No. of Sites	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	137	4.4%	<RL	<RL	196 µg/kg
Urban	63	1.6%	<RL	<RL	34 µg/kg
Mixed	136	6.6%	<RL	<RL	93 µg/kg
Undeveloped	130	6.9%	<RL	<RL	291 µg/kg

Abbreviations:
RL = Reporting limit. Reporting limits for 2,6-dinitrotoluene varied, but did not exceed 50 µg/kg.

For bed sediment, all weights are dry weights.

Most sites were sampled only once. In the case of sites sampled multiple times, USGS used a single sample (the earliest sample with complete data for all analytes) to represent each site in this analysis.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Nowell and Capel, 2003

USGS Stormwater Studies

For the National Highway Runoff Data and Methodology Synthesis, USGS conducted a review of 44 highway and urban runoff studies implemented since 1970 (Lopes and Dionne, 1998). 2,4-and 2,6-DNT were included as analytes in three of these studies. For more background on these studies, see Chapter 2.

All three studies were stormwater studies conducted in major metropolitan areas in connection with National Pollutant Discharge Elimination System (NPDES) permitting. In Maricopa County, Arizona, USGS collected 35 samples from five drainage basins and the City of Phoenix collected an additional 26 samples from seven sites (Lopes *et al.*, 1995). In Colorado Springs, Colorado, 35 samples were collected from five sites (von Guerard and Weiss, 1995). In the Dallas-Fort Worth area of Texas, 182 samples were collected from 26 stormwater drainage basins (Baldys *et al.*, 1998). For both 2,4- and 2,6-DNT, the reporting limit was 5 µg/L in all three studies. Not all samples were monitored for every contaminant. None of the three studies found any detections of 2,4- or 2,6-DNT.

7.3.3 Drinking Water Occurrence

Nationally representative data on 2,4- and 2,6-DNT occurrence in drinking water have been collected by large and small public water systems in accordance with EPA's first Unregulated Contaminant Monitoring Regulation (UCMR 1). For details on UCMR 1, see Chapter 2 and USEPA (2006b).

UCMR 1

UCMR 1 monitoring was conducted primarily between 2001 and 2003, though some results were not collected and reported until as late as 2005. As List 1 contaminants, 2,4- and 2,6-DNT were scheduled to be monitored by all large community water systems (CWSs) and non-transient non-community water systems (NTNCWSs) and a statistically representative sample of qualifying small CWSs and NTNCWSs. The data presented in this report reflect UCMR 1 analytical samples submitted and quality-checked under the regulation as of July 2005. 2,4- and 2,6-Dinitrotoluene data were collected and submitted by 797 (99.6 percent) of the 800 small systems selected for the small system sample and 3,069 (99.0 percent) of the 3,100 large systems defined as eligible for the UCMR 1 large system census. Data for each contaminant have been analyzed at the level of simple detections (at or above the minimum reporting level, ≥ MRL, or ≥ 2 µg/L). Since the health reference level (HRL) of 0.05 µg/L is less than the MRL, the data are not analyzed at the level of the HRL or half the HRL.

EPA set the MRL for UCMR 1 contaminants based on the capability of analytical methods, not anticipated health levels. For many UCMR 1 contaminants, including 2,4- and 2,6-Dinitrotoluene, the MRL was determined by multiplying by 10 the least sensitive method's minimum detection limit, or, when available, multiplying by 5 the least sensitive method's estimated detection limit (USEPA, 2000). MRLs were set approximately an order of magnitude higher than detection limits to ensure consistency, accuracy, and reproducibility of results. The

MRL for 2,4- and 2,6-dinitrotoluene is within the risk range of 10^{-6} to 10^{-4} used by EPA to evaluate carcinogens (see Section 2.1.1).

Results of the analysis are presented in the following four exhibits (Exhibit 7-7, 7-8, 7-9, and 7-10). Among small systems, there were no detections of 2,4- or 2,6-dinitrotoluene. Among large systems, one had a detection of 2,4-dinitrotoluene; this surface water system represented 0.03% of large systems and 0.02% of the population served by them (approximately 38,000 people). The concentration of the single detection was 333 µg/L. No 2,6-dinitrotoluene detections were reported from large systems.

Exhibit 7-7: Summary UCMR 1 Occurrence Statistics for 2,4-Dinitrotoluene in Small Systems (Based on Statistically Representative National Sample of Small Systems)

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,251		--
Percent of Samples with Detections	0.00%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	0.05 µg/L		--
Minimum Reporting Level (MRL)	2 µg/L		--
Maximum Concentration of Detections	< MRL		--
99th Percentile Concentration of Detections	< MRL		--
Median Concentration of Detections	< MRL		--
Total Number of PWSs	797		60,414
Number of GW PWSs	590		56,072
Number of SW PWSs	207		4,342
Total Population	2,760,570		45,414,590
Population of GW PWSs	1,939,815		36,224,336
Population of SW PWSs	820,755		9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	0	0.00%	0

1. PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 7-8: Summary UCMR 1 Occurrence Statistics for 2,4-Dinitrotoluene in Large Systems (Based on the Census of Large Systems)

Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	30,350	
Percent of Samples with Detections	0.003%	
99th Percentile Concentration (all samples)	< MRL	
Health Reference Level (HRL)	0.05 µg/L	
Minimum Reporting Level (MRL)	2 µg/L	
Maximum Concentration of Detections	333 µg/L	
99th Percentile Concentration of Detections	333 µg/L	
Median Concentration of Detections	333 µg/L	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	3,069 1,375 1,694	
Total Population Population of GW PWSs Population of SW PWSs	223,361,341 53,303,000 170,058,341	
Occurrence by System	**Number**	**Percentage**
PWSs (GW & SW) with Detections (≥ MRL) GW PWSs with Detections SW PWSs with Detections	1 0 1	0.03% 0.00% 0.06%
Occurrence by Population Served		
Population Served by PWSs with Detections Pop. Served by GW PWSs with Detections Pop. Served by SW PWSs with Detections	37,811 0 37,811	0.02% 0.00% 0.02%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 7-9: Summary UCMR 1 Occurrence Statistics for 2,6-Dinitrotoluene in Small Systems (Based on Statistically Representative National Sample of Small Systems)

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,251		--
Percent of Samples with Detections	0.00%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	0.05 µg/L		--
Minimum Reporting Level (MRL)	2 µg/L		--
Maximum Concentration of Detections	< MRL		--
99th Percentile Concentration of Detections	< MRL		--
Median Concentration of Detections	< MRL		--
Total Number of PWSs	797		60,414
Number of GW PWSs	590		56,072
Number of SW PWSs	207		4,342
Total Population	2,760,570		45,414,590
Population of GW PWSs	1,939,815		36,224,336
Population of SW PWSs	820,755		9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 7-10: Summary UCMR 1 Occurrence Statistics for 2,6-Dinitrotoluene in Large Systems (Based on the Census of Large Systems)

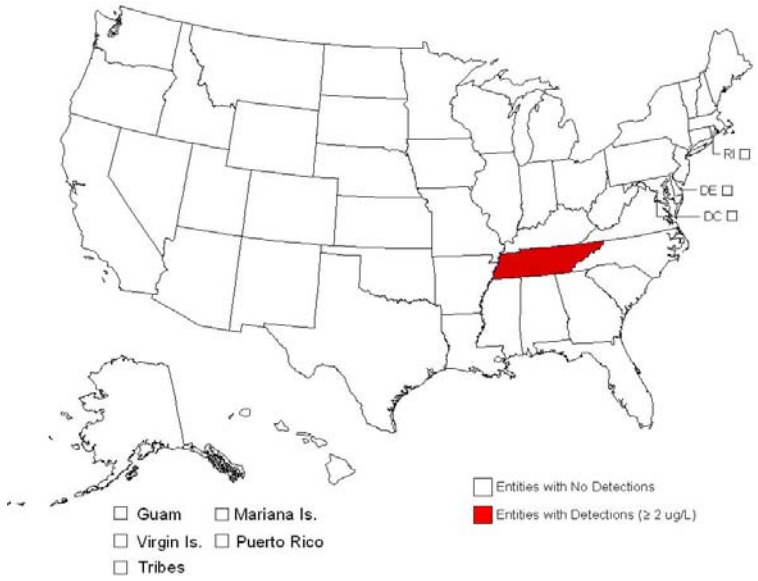
Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	30,351	
Percent of Samples with Detections	0.00%	
99th Percentile Concentration (all samples)	< MRL	
Health Reference Level (HRL)	0.05 µg/L	
Minimum Reporting Level (MRL)	2 µg/L	
Maximum Concentration of Detections	< MRL	
99th Percentile Concentration of Detections	< MRL	
Median Concentration of Detections	< MRL	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	3,069 1,375 1,694	
Total Population Population of GW PWSs Population of SW PWSs	223,361,341 53,303,000 170,058,341	
Occurrence by System	**Number**	**Percentage**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%
Occurrence by Population Served		
Population Served by PWSs with Detections	0	0.00%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

2,4-Dinitrotoluene was only detected in one sample at or above the MRL of 2 µg/L in all of the UCMR 1 sampling. This single detection was in a surface water sample taken in the State of Tennessee (see Exhibit 7-11). Since only one system detected the contaminant at or above the MRL, no further spatial analysis of this contaminant is presented.

Exhibit 7-11: Geographic Distribution of 2,4-Dinitrotoluene in UCMR 1 Monitoring – States With At Least One Detection At or Above the MRL (≥ 2 µg/L)



Summary Analysis of Combined Large and Small System UCMR 1 Data

The UCMR 1 data indicate that 1 of the 3,866 public water systems (PWSs) sampled (or 0.03 percent) detected 2,4-DNT at the MRL of 2 µg/L, affecting 0.02 percent of the population served (or 38,000 people from 226 million). None of the 3,866 PWSs sampled (serving 226 million) detected 2,6-DNT at the MRL of 2 µg/L.

7.4 Technology Assessment

7.4.1 Analytical Methods

EPA evaluated the availability of analytical methods for all of the unregulated contaminants considered for UCMR 1 (64 FR 50556). Sources for these methods include publications by EPA and by voluntary consensus standard organizations such as the American Society for Testing and Materials (ASTM), the Association of Official Analytical Chemists (AOAC), and the American Public Health Association (APHA).

2,4- and 2,6-Dinitrotoluene are UCMR 1 List 1 contaminants that can be detected in drinking water by EPA Method 525.2. This method was approved for monitoring 2,4- and 2,6-

dinitrotoluene in 1999 (64 FR 50556). EPA Method 525.2 relies on capillary column gas chromatography (GC) to separate the method analytes, and uses mass spectrometry (MS) for detection. A full description of this method can be found in EPA's *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III* (USEPA, 1995a).

EPA Method 525.2

In EPA Method 525.2 (Revision 2.0), "*Determination of Organic Compounds in Drinking Water by Liquid-Solid Phase Extraction and Capillary Column Gas Chromatography/Mass Spectrometry (GC/MS)*," organic compound analytes are first extracted from a water sample by passing the water through a liquid-solid extraction (LSE) disk or cartridge containing a solid matrix with a chemically bonded C_{18} organic phase. The organic compounds are eluted from the LSE cartridge or disk with small volumes of ethyl acetate and methylene chloride. These solvent extracts are concentrated further by evaporation of some of the solvent. An aliquot of the extract is injected into a gas chromatograph with a high resolution fused silica capillary column to separate the components. The analytes are transferred from the capillary column to the mass spectrometer and identified by comparing measured mass spectra and retention times to reference spectra and retention times. The concentration of each component is assessed by comparing the mass spectrometry response of the compound's quantitation ion to the response of the internal standard's quantitation ion (USEPA, 1995b). Mass spectrometry is advantageous as a detection method because it reports comparatively few false positive results.

The MDL for Method 525.2 is reported to range from 0.072 to 0.24 µg/L for 2,4-dinitrotoluene and from 0.054 to 0.2 µg/L for 2,6-dinitrotoluene, depending upon the extraction media used (USEPA, 1995b). The average recovery is reported to range from 59 to 119 percent for 2,4-dinitrotoluene and 56 to 121 percent for 2,6-dinitrotoluene, depending upon the method option used (USEPA, 1995b).[2]

Anecdotal reports from laboratories performing Method 525.2 for the analysis of dinitrotoluenes for UCMR 1 have indicated that they are having difficulty obtaining satisfactory recoveries for these compounds, and are therefore having difficulty meeting the quality control (QC) requirements of the method. A preliminary investigation indicates that this may be related to manufacturing changes to the LSE sorbent disks distributed by a major vendor. Satisfactory recoveries of these analytes are still obtained using LSE sorbent disks manufactured by other vendors or by using the method's cartidge option. In the event that regulatory action needs to be

[2] The Method Detection Limit (MDL) is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon the precision of a series of replicate measurements of the analyte at low concentrations. The MDL incorporates estimates of the accuracy of the determination. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

The average recovery is the fraction or percent concentration of a target analyte determined relative to the true or expected concentration from a sample containing a known amount of the target analyte. (This can result in apparent recovery values greater than 100 percent.)

taken, a review of the suitability of Method 525.2 for compliance monitoring will be needed. It is also possible that the dinitrotoluenes could be incorporated into other existing EPA drinking water methods available for compliance monitoring.

7.4.2 Treatment Technologies

Treatment technology status does not influence the determination of whether or not a contaminant should be regulated. However, treatment technologies must be readily available before a contaminant can be regulated with a national primary drinking water regulation (NPDWR). Potential treatment technologies for removing 2,4- and 2,6-dinitrotoluene include activated carbon and air stripping.

Granular activated carbon (GAC) treatment removes contaminants via the physical and chemical process of sorption: the contaminants attach to the carbon surface as water passes through the carbon bed. Activated carbon has a large sorption capacity for many water impurities, including synthetic organic chemicals, taste- and odor-causing compounds, and some species of mercury.

Adsorption capacity is typically represented by the Freundlich isotherm constant, with higher Freundlich (K) values indicating greater sorption potential. Activated carbon is considered to be cost-effective for removing a particular contaminant if the Freundlich (K) value of the contaminant is above 200 $\mu g/g\ (L/\mu g)^{1/n}$ (Speth *et al.*, 2001). Dobbs and Cohen (1980 as cited in Speth *et al.*, 2001) report that the Freundlich (K) values for 2,4- and 2,6-dinitrotoluene are 17,200 $\mu g/g\ (L/\mu g)^{1/n}$ and 15,900 $\mu g/g\ (L/\mu g)^{1/n}$, respectively, which suggests that GAC is a promising treatment option for both.

Air stripping involves the continuous contact of air with the water being treated, allowing dissolved volatile contaminants to transfer from the source water to the air. Systems often consist of a large column (or tower) filled with molded plastic or ceramic packing material. As the water flows along the column, air is forced counter-current through the water. The packing material increases the area of air-liquid interface, enhancing mass transfer. After contact, the air is vented to an additional treatment device that safely contains or destroys the contaminant.

The Henry's Law constant is commonly used to indicate the tendency of a contaminant to partition from water to air. A larger Henry's constant indicates a greater equilibrium concentration of the contaminant in the air. Thus, contaminants with larger Henry's constants are more efficiently removed by air stripping. A compound is generally considered amenable to air stripping if it has a Henry's law constant above that of dibromochloropropane (0.003 mol/mol) or ethylene dibromide (0.013 mol/mol) (Speth *et al.*, 2001). Speth *et al.* (2001) compiled Henry's Law constants, both calculated by the authors and reported in the literature, for Contaminant Candidate List (CCL) compounds. These authors report Henry's Law constants of 0.22 mol/mol and 7.4 mol/mol for 2,6-dinitrotoluene, and 0.000046 mol/mol for 2,4-dinitrotoluene. These values suggest that air stripping is a promising treatment option for 2,6-dinitrotoluene, but that is not likely to be viable for 2,4-dinitrotoluene (Speth *et al.*, 2001).

7.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate 2,4- or 2,6-DNT with a national primary drinking water regulation (NPDWR). Because 2,4- and 2,6-DNT appear to occur infrequently at levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. While 2,4-DNT was detected once at a minimum reporting level that is within the 10^{-4} to the 10^{-6} cancer risk range, 2,6-DNT was not detected at this same level in any of the PWSs monitored under the UCMR 1.

EPA recognizes that 2,4- and 2,6-DNT are listed as probable human carcinogens. For this reason, the Agency encourages those States with public water systems that may have either 2,4- or 2,6-DNT above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency's original Health Advisories for 2,4 and 2,6-DNT were developed for military installations. Because the Agency recognizes that 2,4 and 2,6-DNT may still be found at some military sites, the Agency has updated the Health Advisories to reflect recent health effects publications. The Health Advisories are available for review in the docket. The updated Health Advisories will provide information to any States with public water systems that may have either 2,4- or 2,6-DNT above the HRL.

The Agency's preliminary regulatory determination for these contaminants is presented formally in the *Federal Register*.

7.6 References

Agency for Toxic Substances and Disease Registry (ATSDR). 1998. *Profile for 2,4- and 2,6-dinitrotoluene*. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services. Available on the Internet at: http://www.atsdr.cdc.gov/toxprofiles/tp109.html.

Baldys, S, III, T.H. Raines, B.L. Mansfield, and J.T. Sandlin. 1998. *Urban stormwater quality, event mean concentrations, and estimates of stormwater pollutant loads, Dallas-Fort Worth area, Texas, 1992–93.* U.S. Geological Survey Water-Resources Investigation Report 98-4158, 51 p.

Bidleman, T.F. 1988. Atmospheric processes. Wet and dry deposition of organic compounds are controlled by their vapour-article partitioning. *Environmental Science and Technology*. 22:361-367. (As cited in HSDB, 2004).

Bradley, P.M., F.H. Chapelle, and J.E. Landmeyer. 1994. Microbial transformation of nitroaromatics in surface soils and aquifer materials. *Applied and Environment Microbiology*. 60:2170-2175. (As cited in ATSDR, 1998).

Dobbs, R.A. and J.M. Cohen. 1980. *Carbon Adsorption Isotherms for Toxic Organics*. EPA Report 600-8-80-023. Cincinnati, OH: Municipal Environmental Research Laboratory, USEPA. April, 1980. (As cited in Speth *et al.*, 2001).

Ellis, H.V., J.H. Hagensen, J.R. Hodgson, *et al.* 1979. *Mammalian toxicity of munitions compounds. Phase BI: Effects of lifetime exposure. Part I: 2,4-Dinitrotoluene.* Final report no. 7. (As cited in USEPA, 1992).

Ellis, H.V., C.B. Hong, C.C. Lee, J.C. Dacre, and J.P. Glennon. 1985. Subchronic and chronic toxicity studies of 2,4-dinitrotoluene. Part I: Beagle dog. *Journal of the American College of Toxicology*. 4:233-242. (As cited in USEPA, 1992).

Etnier, E.L. 1987. *Water quality criteria for 2,4-dinitrotoluene and 2,6-dinitrotoluene*. Oak Ridge, TN: Oak Ridge National Laboratory. U.S. Army Medical Research and Development Command. Project order no.84PP4845. (As cited in ATSDR, 1998).

Grant. C.L., T.F. Jenkins, K.F. Meyers, and E.F. McCormick. 1995. Holding-time estimates for soils containing explosive residues: comparison of fortification vs. field contamination. *Journal of Environmental Toxicology and Chemistry*. 14:1865-1874. (As cited in ATSDR, 1998).

Gruener, N. 1976. Ontogenetic development of NADH-dependent methemoglobin reductase in erythrocytes of man and rat. *Journal of Toxicology and Environmental Health*. 1:787-791. (As cited in ATSDR, 1998).

Hansch, C., A.J. Leo, and D. Hoekman. 1995. *Exploring QSAR – Hydrophobic, Electronic, and Steric Constants*. Washington, DC: American Chemical Society. (As cited in HSDB, 2004).

Hazardous Substance Release/Health Effects Database (HazDat). 1998. Agency for Toxic Substances and Disease Registry (ATSDR), Atlanta, GA. (As cited in ATSDR, 1998).

Hazardous Substances Data Bank (HSDB). 2004. Searches for 2,4-dinitrotoluene and 2,6-dinitrotoluene. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB, accessed November 1, 2004.

Hong, C.B., H.V. Ellis, C.C. Lee, H. Sprinz, J.C. Dacre, and J.P. Glennon. 1985. Subchronic and chronic toxicity studies of 2,4-dinitrotoluene. Part III. CD-1 Mice. *Journal of the American College of Toxicology*. 4(4)257-269. (As cited in ATSDR, 1998)

Howard, P.H. 1990. *Handbook of Environmental Fate and Exposure Data for Organic Chemicals*. Volume 1. Chelsea, MI: Lewis Publishers, Inc.

Jenkins, T.F., P.G. Thorne, E.F. McCormick, and K.F. Myers. 1995. *Preservation of Water Samples Containing Nitroaromatics and Nitramines.* Special Report 95-16, U.S. Army Corps of Engineers, Cold Regions Research and Engineering Laboratory. Available on the Internet at: http://www.crrel.usace.army.mil/techpub/CRREL_Reports/reports/SR95_16.pdf.

Lee, C.C., J.V. Dilley, J.R. Hodgson, D.O. Helton, W.J. Wiegand, D.N. Roberts, B.S. Andersen, B.N. Halfpap, L.D. Kurtz, and N. West. 1975. *Mammalian toxicity of munition compounds: Phase I. Acute oral toxicity, primary skin and eye irritation, dermal sensitization, and disposition and metabolism.* (Unpublished study, as cited in ATSDR, 1998).

Lee C.C., H.V. Ellis III, J.J. Kowalski, J.R. Hodgsen, R.D. Short, J.C. Bhandari, T.W. Reddig, and J.L. Minor. 1976. *Mammalian toxicity of munitions compounds. Phase II: Effects of multiple doses. Part III: 2,6-Dinitrotoluene.* (Unpublished study, as cited in USEPA, 1992).

Lee, C.C., H.V. Ellis III, J.J. Kowalski, J.R. Hodgson, and S.W. Hwang. 1978. *Mammalian toxicity of munitions compounds. Phase II: Effects of multiple doses. Part II: 2,4-Dinitrotoluene.* (Unpublished study, as cited in ATSDR, 1998).

Lee, C.C., C.B. Hong, H.V. Ellis III, J.C. Dacre, and J.P. Glennon. 1985. Subchronic and chronic toxicity studies of 2,4-dinitrotoluene. Part II. CD rats. *Journal of the American College of Toxicology*. 4:243-256. (As cited in ATSDR, 1998).

Lide, D.R. (ed.). 1999. *CRC Handbook of Chemistry and Physics*. 79th ed. Boca Raton, FL: CRC Press Inc. (As cited in HSDB, 2004).

Lopes, T.J., K.D. Fossum, J.V. Phillips, and J.E. Monical. 1995. *Statistical summary of selected physical, chemical, and microbial characteristics, and estimates of constituent loads in urban stormwater, Maricopa County, Arizona.* U.S. Geological Survey Water-Resources Investigations Report 94-4240.

Lopes, T.J. and S.G. Dionne. 1998. *A Review of Semivolatile and Volatile Organic Compounds in Highway Runoff and Urban Stormwater.* U.S. Geological Survey Open-File Report 98-409, 67 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr98-409.pdf.

Lyman W.J., W.J. Reehl, and D.H. Rosenblatt. 1982. *Handbook of Chemical Property Estimation Methods*. New York, NY: McGraw-Hill Book Co. (As cited in Howard, 1990).

Mabey, W.R., J.H. Smith, R.T. Podoll, H.L. Johnson, T. Mill, T.W. Chou, J. Gates, I.W. Partridge, H. Jaber and D. Vandenberg. 1982. *Aquatic fate process data for organic priority pollutants*. EPA Report 440-4-81-014, 239-243. (As cited in ATSDR, 1998).

Nakagawa, Y., K. Izumi, N. Oikawa, T. Sotomatsu, M. Shigemura, and T. Fujita. 1992. Analysis and prediction of hydrophobicity parameters of substituted acetanilides, benzamides and related aromatic compounds. *Environmental Toxicology and Chemistry*. 11:901-916. (As cited in HSDB, 2004).

National Institute of Standards and Technology (NIST). 2001. *NIST Chemistry WebBook*. Search for 2,4 dinitroluene and 2,6 dinitrotoluene. Available on the Internet at: http://webbook.nist.gov/. Accessed June 29, 2001.

Nowell, L. and P. Capel. 2003. "Semivolatile Organic Compounds (SVOC) in Bed Sediment from United States Rivers and Streams: Summary Statistics; Preliminary Results of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/svoc/SVOC-SED_2001_Text.html. Accessed August 25, 2004. Last updated May 12, 2003.

Pella, P.A. 1977. Measurement of the vapor pressures of TNT, 2,4-DNT, 2,6-DNT, and EGDN. *Journal of Chemical Thermodynamics*. 9(4):301-305. (As cited in HSDB, 2004).

Price, C.J., R.W. Tyl, T.A. Marks., L.L. Paschke, T.A. Ledoux, and J.R. Reel. 1985. Teratologic evaluation of dinitrotoluene in the Fischer 344 rat. *Fundamental and Applied Toxicology*. 5:948-961. (As cited in USEPA, 1992).

Smith, J.H., D. Mackay and C. Ng. 1983. Volatilization of pesticides from water. *Residue Reviews*. 85:73-88. (As cited in Howard, 1990).

Society for German Chemists (SGC). 1987. *Dinitrotoluenes: BUA Substance Report 12*. The Advisory Board for Environmentally Significant Hazardous Wastes (BUA) of the Society of German Chemists, eds. Weinheim, Fed Rep of Germany: VCH Verlagsgesellschaft. (As cited in Howard, 1990).

Spanggord, R.J., W.R. Mabey, T. Mill, T.W. Chou, J.H. Smith, and S. Lee. 1980. Environmental Fate Studies on Certain Munition Wastewater Constituents. Final Report, Phase I - Literature Review. DAMD 17-78-C8081. (As cited in HSDB, 2004).

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

United States Environmental Protection Agency (USEPA). 1980. *Ambient Water Quality Criteria Document: Dinitrotoluene*. EPA Report 440/5-80-045. (As cited in HSDB, 2004).

USEPA. 1990. United States Environmental Protection Agency. Integrated Risk Information System (IRIS) for 2,4-/2,6-Dinitrotoluene Mixture. Cincinnati, OH. Available at: http://www.epa.gov/iris/subst/0397.htm.

USEPA. 1992. *2,4-& 2,6-Dinitrotoluene (DNT) Health Advisory.* EPA Report 820-R-92-002. April 1992.

USEPA. 1995a. *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III*. EPA Report 600-R-95-131. August, 1995.

USEPA. 1995b. Method 525.2−Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry (GC/MS). Revision 2.0. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III.* EPA Report 600-R-95-131. August, 1995.

USEPA. 2000. *Unregulated Contaminant Monitoring Regulation Analytical Methods and Quality Control Manual*. EPA Report 815-R-00-006.

USEPA. 2006a. TRI Explorer: Trends. Searches for 2,4-dinitrotoluene, 2,6-dinitrotoluene, and dinitrotoluene (mixed isomers). Available on the Internet at: http://www.epa.gov/triexplorer/trends.htm. Accessed February 8, 2006. Last modified June 8, 2005.

USEPA. 2006b. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

von Guerard, P. and W.B. Weiss. 1995. *Water quality of storm runoff and comparison of procedures for estimating storm-runoff loads, volume, event-mean concentrations, and the mean load for a storm for selected properties and constituents for Colorado Springs, southeastern Colorado, 1992*. U.S. Geological Survey Water-Resources Investigations Report 94-4194.

Chapter 8:
EPTC

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

s-Ethyl dipropylthiocarbamate (EPTC), a synthetic organic compound (SOC), is a thiocarbamate herbicide used to control weed growth during the pre-emergence and early post-emergence stages of weed germination. First registered for use in 1958, EPTC is used across the U.S. in the agricultural production of a number of crops, most notably corn, potatoes, dried beans, alfalfa, and snap beans. EPTC is also used residentially on shade trees, annual and perennial ornamentals, and evergreens.

Environmental fate data indicate that EPTC would not be persistent under most environmental conditions. Volatilization into the atmosphere and degradation by soil organisms appear to be the primary dissipation routes. EPTC has a low affinity for binding to the soil so the potential to leach to ground water does exist. If EPTC reaches ground water, volatilization is less likely to occur.

In subchronic and chronic studies performed in both rats and dogs, EPTC exposure produced dose-related increases in the incidence and severity of cardiomyopathy, a disorder of the heart muscle, and degenerative effects (neuronal and/or necrotic degeneration) in the central and peripheral nervous system. Based on a no-observed-adverse-effect level (NOAEL) of 2.5 mg/kg/day from a study that found cardiomyopathy at higher doses, EPA derived a reference dose (RfD) of 0.025 mg/kg/day for EPTC. This value was calculated using an uncertainty factor of 100 for inter- and intraspecies differences. The Agency derived the health reference level (HRL) for EPTC using the RfD of 0.025 mg/kg/day and a 20 percent relative source contribution. The HRL is calculated to be 0.175 mg/L or 175 µg/L.

The Agency used long-term studies in mice and rats and short-term studies of mutagenicity to evaluate the potential for EPTC carcinogenicity. Based on these data and using EPA's 1999 Guidelines for Carcinogen Risk Assessment, EPA considers EPTC unlikely to be carcinogenic to humans.

Available data do not suggest increased pre- or post-natal sensitivity of children and infants to EPTC exposure.

Estimates of EPTC usage in the United States suggest a decline from approximately 17 to 21 million pounds in 1987 to approximately 7 to 9 million pounds in 1999. Toxic Release Inventory (TRI) data from 1995 to 2003 indicate that most on-site industrial releases of EPTC tend to be releases to air and underground injections. Surface water discharges are minimal in comparison.

Data on the ambient occurrence of EPTC are available from the first monitoring cycle (1992-2001) of the United States Geological Survey's (USGS's) National Ambient Water Quality Assessment (NAWQA) program. While USGS detected EPTC in both surface and ground waters, in no land use setting did the 95th percentile concentration of EPTC exceed 0.018 µg/L. The estimated maximum surface water concentration, 29.6 µg/L (from a mixed land use setting), and the maximum ground water concentration, 0.45 µg/L (from an agricultural setting), are both less than the EPTC HRL and ½ the HRL.

To determine the extent of EPTC contamination in drinking water, EPA included EPTC as an analyte in the first Unregulated Contaminant Monitoring Regulation (UCMR 1). None of the 3,866 public water systems (PWSs) sampled (serving a total population of 226 million) had detects of EPTC at or above the minimum reporting level (MRL) of 1 µg/L. Hence, these data indicate that no occurrence and exposure is expected in drinking water at levels greater than the HRL (175 µg/L), or even ½ the HRL (87.5 µg/L).

EPA also evaluated sources of supplemental information on EPTC occurrence in drinking water. The National Pesticide Survey (NPS) collected samples from approximately 1,300 community water systems and rural drinking water wells between 1988 and 1990. EPTC was not detected using a minimum reporting limit of 0.15 µg/L. The Pesticides in Ground Water Database indicates that EPTC was found in 2 of 1,752 ground water wells that were sampled in 10 States. Both contaminated wells were in Minnesota. The detected concentrations ranged from 0.01 to 0.33 µg/L. No detections exceeded the HRL or ½ the HRL.

The Agency has made a preliminary determination not to regulate EPTC with a national primary drinking water regulation (NPDWR). Because EPTC does not appear to occur at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

a.i.	Active Ingredient
AOAC	Association of Official Analytical Chemists
APHA	American Public Health Association
ASTM	American Society for Testing and Materials
CAS	Chemical Abstracts Service
CCL 2	Second Contaminant Candidate List
ChE	Cholinesterase
CWS	Community Water System
EPTC	s-Ethyl dipropylthiocarbamate
ESO	EPTC sulfoxide
FQPA	Food Quality Protection Act
GAC	Granular Activated Carbon
GC	Gas Chromatography
GC/MS	Gas Chromatography with Mass Spectrometry
HRL	Health Reference Level
LSE	Liquid-Solid Extraction
MDL	Method Detection Limit
MRL	Minimum Reporting Level
MTBE	Methyl tertiary-butyl ether
NAWQA	National Water Quality Assessment
NCFAP	National Center for Food and Agricultural Policy
NOAEL	No Observed Adverse Effect Level
NPD	Nitrogen-Phosphorus Detector
NPDWR	National Primary Drinking Water Regulation
NPS	National Pesticide Survey
NTNCWS	Non-Transient Non-Community Water System
OPP	Office of Pesticide Programs
PGWDB	Pesticides in Ground Water Database
RfD	Reference Dose
RL	Reporting Limit
RO	Reverse Osmosis
SOC	Synthetic Organic Compound
TRI	Toxics Release Inventory
UCMR 1	First Unregulated Contaminant Monitoring Regulation
USGS	United States Geological Survey

8 EPTC

8.1 Definition

Dipropylthiocarbamic Acid S-Ethyl Ester (EPTC) is a thiocarbamate (a carbamate in which the -CO- group has been replaced by a -CS- group) herbicide. It is included in the category of synthetic organic compounds (SOCs). Synonyms include: S-ethyl dipropyl-thiocarbamate, R-1608, FDA 1541, and Eptam (Windholz, 1983). Additional trade names include Alirox, Eradicane, Eradicane Extra, Genep, Genep Plus, and Shortstop. It has no predominant isomers. EPTC's Chemical Abstracts Service (CAS) registry number is 759-94-4.

8.1.1 Properties and Sources

EPTC is a colorless or yellow liquid with a characteristic odor. It is a synthetic product and does not occur naturally. The predominant use of EPTC is as a selective herbicide. EPTC is used for control of annual grassy weeds, perennial weeds, and some broadleaf weeds in the cultivation of beans, forage legumes, potatoes, corn, and sweet potatoes. EPTC is produced in several ways, but commonly by the reaction of dipropylamine with ethyl chlorothioformate (HSDB, 2004). Some physical and chemical properties of EPTC are listed in Exhibit 8-1.

Exhibit 8-1: Physical and Chemical Properties of EPTC

Identification	
CAS number	759-94-4
Molecular Formula	$C_9H_{19}NOS$
Physical and Chemical Properties	
Boiling Point	127 °C at 20 mm Hg [1]
Melting Point	-----
Molecular Weight	189.31 g/mol [1]
Log K_{oc}	2.23 - 2.45 [2]
Log K_{ow}	3.21 [3]
Water Solubility	367 mg/L at 25 °C [4]
Vapor Pressure	2.4×10^{-2} mm Hg at 25 °C [5]
Henry's Law Constant	1.6×10^{-5} atm-m^3/mol [2] 9.8×10^{-4} (dimensionless), predicted [6] 6.5×10^{-4} (dimensionless), from literature [6]
Freundlich Isotherm Constant (K)	79,500 (µg/g)(L/µg)$^{1/n}$ [6]

[1] *Tomlin, 1997 (as cited in HSDB, 2004)*

[2] *HSDB, 2004*

[3] *Hansch et al., 1995 (as cited in HSDB, 2004)*

[4] *Yalkowsky and Dannenfelser, 1992 (as cited in HSDB, 2004)*

[5] *USDA, 2000 (as cited in HSDB, 2004)*

[6] *Speth et al., 2001*

8.1.2 Environmental Fate and Behavior

Microbial degradation and volatilization are the primary environmental pathways of EPTC in soil. EPTC is readily lost from soil surfaces by volatilization if not incorporated into the soil upon application. Terrestrial field dissipation studies report soil half-lives between 2 to 18.8 days. Judging by its water solubility of 367 mg/L and its low affinity for binding to soil, EPTC also has a moderate potential to leach into ground water during this short window. Abiotic hydrolysis, direct photolysis, and photodegradation are not major degradation routes. EPTC is somewhat more persistent in anaerobic soils than in aerobic soils. (USEPA, 1999a).

EPTC is likely to persist longer in ground waters than in surface waters due to its relatively high volatility (USEPA, 1999a). Microbial degradation is also expected to be a significant pathway in aquatic environments, but there have been no studies to confirm this (USEPA, 1999a).

EPTC in the atmosphere is expected to remain primarily in the vapor phase. Atmospheric EPTC may degrade by reaction with photochemically produced hydroxyl radicals and may also be subject to wet deposition, potentially contaminating nonagricultural sites and surface waters (USEPA, 1999a).

The primary environmental degradates of EPTC are EPTC sulfoxide (ESO) and dipropylamine. ESO is formed during oxidation of EPTC, the first step of the compound's breakdown. Subsequent sulfur and carbon oxidation produces dipropylamine. Other degradation pathways have also been proposed (USEPA, 1999a). Half-lives for ESO and dipropylamine in soil have been estimated at 13-14 days and 7 days, respectively. While environmental fate data for EPTC degradates are limited, available data suggest that ESO and dipropylamine may be less mobile than the parent compound (USEPA, 1999a).

8.2 Health Effects

In acute animal toxicity studies, EPTC was shown to be moderately toxic via oral and dermal routes and highly toxic via inhalation exposures. EPTC is a reversible cholinesterase (ChE) inhibitor. Similar to other thiocarbamates, it does not produce a consistent ChE inhibition profile. There was no consistent pattern observed in any of the toxicity studies with regard to species, duration of treatment, or the type of ChE enzyme measured. Typically, studies showed inhibition of plasma ChE with dose-related decreases in red blood cell and brain ChE activity. Some studies have shown that brain ChE activity was inhibited without any effect on either plasma or erythrocyte ChE activities. Other studies illustrated erythrocyte ChE inhibition with no effect on either plasma or brain ChE (USEPA, 1999a). In a primary eye irritation study in rabbits, technical grade EPTC was shown to be slightly irritating (USEPA, 1999a).

In subchronic and chronic studies performed in both rats and dogs, there was a dose-related increase in the incidence and severity of cardiomyopathy, a disorder of the heart muscle (Mackenzie, 1986 as cited in USEPA, 1999a; USEPA, 1999a). An increase in the incidence and severity of degenerative effects (neuronal and/or necrotic degeneration) in both the central and peripheral nervous system was observed in rats and dogs following exposure to EPTC (USEPA, 1999a).

EPA derived a reference dose (RfD) of 0.025 mg/kg/day for EPTC (USEPA, 1990a; USEPA, 1999a). This value was calculated using a "no-observed-adverse-effect level" (NOAEL) of 2.5 mg/kg/day from a study by Mackenzie (1986 as cited in USEPA, 1999a). An uncertainty factor of 100 was applied for inter- and intraspecies differences. The critical effect associated with the RfD is cardiomyopathy (disease of the heart muscle). In the reregistration of EPTC, the application of a ten-fold Food Quality Protection Act (FQPA) factor was recommended in order to be protective against residential exposures of infants and children. The Agency derived the health reference level (HRL) for EPTC using the RfD of 0.025 mg/kg/day and a 20 percent relative source contribution. The HRL is calculated to be 0.175 mg/L or 175 µg/L.

The Agency used long-term studies in mice and rats and short-term studies of mutagenicity to evaluate the potential for carcinogenicity (USEPA, 1990a). Based on these data and using EPA's 1999 Guidelines for Carcinogen Risk Assessment, EPTC is not likely to be carcinogenic to humans (USEPA, 1999b).

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. Data do not suggest increased pre- or post-natal sensitivity of children and infants to EPTC exposure. In animal studies, adverse developmental effects (i.e., decreased fetal body weight and decreased litter size) were only seen at doses that were toxic to the mother (USEPA, 1999a). Results from both developmental and reproductive studies indicate that there are only minimal adverse effects. The behavior patterns of children that lead to heightened opportunities for exposure in the indoor environment and the need for a developmental neurotoxicity study lead the Office of Pesticide Programs (OPP) to recommend the application of a ten-fold FQPA factor for EPTC. However, EPA did not apply this factor in the screening analysis because it does not apply to programs other than the pesticide registrations.

8.3 Occurrence and Exposure

8.3.1 Use and Environmental Release

EPTC is a thiocarbamate herbicide used in the pre-emergence and early post-emergence stages of weed germination to control weed growth. It was first registered for use in the United States in 1958. It is in widespread use across the United States in agricultural production of a number of crops, most notably corn, potatoes, dried beans, alfalfa, and snap beans. EPTC is also used residentially on shade trees, annual and perennial ornamentals, and evergreens. EPTC can be applied as a spray, as a granular formulation, or via chemigation. EPTC was initially manufactured in Hungary and imported into the United States. Currently, Zeneca Ag Products holds registrations for a number of end-use products and is the sole registration for the technical product in the United States (USEPA, 1999a).

According to EPA statistics from 1987 through 1999, EPTC use in the United States has been declining. In 1999, it was the nineteenth most commonly used active ingredient (a.i.) in U.S. agriculture, down from eighth in 1987 and twelfth in 1993 (USEPA, 2002). According to one analysis, the annual total domestic usage of EPTC between 1987 and 1996 averaged approximately 20 million pounds a.i. for almost 6 million acres treated (USEPA, 1999a). In 2002, EPA concluded that the usage range in 1999 had fallen to between 7 and 9 million pounds a.i., down from 17 to 21 million pounds a.i. in 1987 and 10 to 15 million pounds a.i. in 1993 (USEPA, 2002).

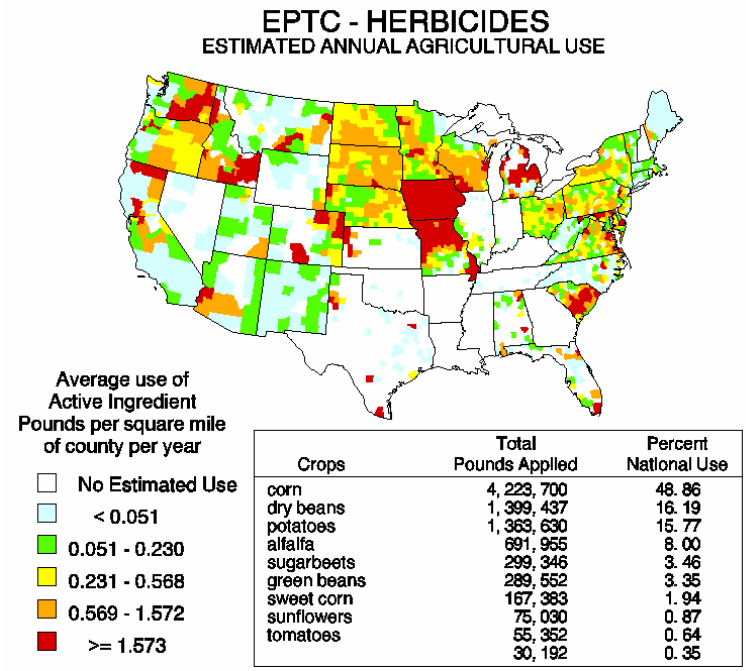
The National Center for Food and Agricultural Policy (NCFAP) estimates of national agricultural ETPC use confirm a decline during the 1990s. According to NCFAP, around 1992 approximately 14.5 million pounds a.i. of EPTC were applied annually to 14 types of crops on 4.0 million acres, and around 1997 approximately 8.8 million pounds a.i. were applied annually to 14 types of crops on 2.6 million acres. NCFAP estimates are based on State-level commercial agriculture usage patterns for the periods 1990-1993 and 1995-1998, and State-level crop acreage for 1992 and 1997 (NCFAP, 2004). For more information on NCFAP pesticide use estimates, see Chapter 2.

The United States Geological Survey (USGS) combined data collected by NCFAP with data from the Census of Agriculture to estimate that 14.1 million pounds of EPTC a.i. per year

were used on approximately 4.0 million agricultural acres in the early 1990s (Thelin and Gianessi, 2000). While USGS has not published national estimates for 1997, an estimate of approximately 8.6 million pounds a.i. can be inferred from the "total pounds applied" and "percent national use" data in the 1997 geographical distribution map (see Exhibit 8-2).

Exhibit 8-2 shows the estimated geographic distribution and intensity of typical annual EPTC use in the United States in the late 1990s. A breakdown of use by crop is also included. The map was created by USGS using State-level data sets on pesticide use rates from 1995-1998 compiled by NCFAP, combined with county-level data on harvested crop acreage obtained from the 1997 Census of Agriculture (USGS, 2004). Due to the nature of the data sources, non-agricultural uses are not reflected on the map and variations in use at the county-level are also not well represented (Thelin and Gianessi, 2000). For background on the USGS pesticide use maps, see Chapter 2. The map indicates that EPTC use is widespread, especially in the East, the Northern Great Plains, and the West.

Exhibit 8-2: Estimated Annual Agricultural Use of EPTC, c. 1997



Crops	Total Pounds Applied	Percent National Use
corn	4,223,700	48.86
dry beans	1,399,437	16.19
potatoes	1,363,630	15.77
alfalfa	691,955	8.00
sugarbeets	299,346	3.46
green beans	289,552	3.35
sweet corn	167,383	1.94
sunflowers	75,030	0.87
tomatoes	55,352	0.64
	30,192	0.35

Source: USGS, 2004

Toxics Release Inventory (TRI) data for EPTC (see Exhibit 8-3) are reported for the years 1995 to 2003 (USEPA, 2006a). Total reported EPTC releases fluctuated widely in the range of thousands of pounds per year during this period. On-site releases were dominated by air emissions and sometimes underground injections. On-site surface water releases did not exceed 300 pounds per year; no land releases were reported. Off-site releases were significant, but declined steadily after 1998. Releases were reported from seven States during the eight-year

period on record. Releases were reported from Alabama, Nebraska, and Louisiana every year or nearly every year. For a discussion of the nature and limitations of TRI data, see Chapter 2.

Exhibit 8-3: Environmental Releases (in Pounds) of EPTC in the United States, 1995-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1995	2,363	291	373	0	9,366	12,393
1996	7,325	2	29	0	590	7,946
1997	2,208	113	9,501	0	2,778	14,600
1998	2,008	115	2,088	0	4,565	8,776
1999	2,574	156	903	0	3,570	7,203
2000	2,034	95	6,083	0	2,798	11,010
2001	2,034	99	1,146	0	1,655	4,934
2002	1,917	98	0	0	708	2,723
2003	1,575	95	0	0	513	2,183

Source: USEPA, 2006a

8.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are the source of most drinking water. Data on the occurrence of EPTC in ambient surface and ground water are available from the National Water Quality Assessment (NAWQA) program of the USGS. For details on this program, see the discussion in Chapter 2. NAWQA data have been analyzed independently by USGS and EPA.

NAWQA National Pesticide Synthesis

Under the NAWQA program, USGS monitored EPTC between 1992 and 2001 in representative watersheds and aquifers across the country. Reporting limits varied but did not exceed 0.002 µg/L.

In surface water (Exhibit 8-4), EPTC was detected at frequencies ranging from 1.64% of samples in undeveloped settings to 4.81% in urban land use settings, 11.88% in mixed land use settings, and 14.11% in agricultural settings. The 95th percentile concentrations were less than the reporting limit in undeveloped and urban settings, 0.009 µg/L in mixed land use settings, and 0.018 µg/L in agricultural settings. The highest concentration, estimated at 29.6 µg/L, was found in a mixed land use setting (Martin *et al.*, 2003).

Exhibit 8-4: USGS National Synthesis Summary of NAWQA Monitoring of EPTC in Ambient Surface Water, 1992-2001

Land Use Type	No. of Samples (and No. of Sites)	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,884 (78)	14.11%	<RL	0.018 µg/L	7.30 µg/L
Mixed	1,000 (47)	11.88%	<RL	0.009 µg/L	29.6 µg/L (E)
Undeveloped	60 (4)	1.64%	<RL	<RL	0.004 µg/L
Urban	892 (33)	4.81%	<RL	<RL	0.038 µg/L

Notes:

RL = Reporting limit. Reporting limits for EPTC varied, but did not exceed 0.002 µg/L.

E = Estimated (outside normal calibration limits)

The USGS Pesticide National Synthesis used one year of data, generally the year with the most sampling results, to represent each site in this analysis. The sampling results were time-weighted, to eliminate bias from more frequent sampling at certain times of year. Detection Frequencies and Percentile Concentrations can be interpreted as representing annual occurrence. For instance, the detection frequency can be thought of as the percent of the year in which detections are found at a typical site in this land use category, and the 95th percentile concentration can be thought of as a concentration that is not exceeded for 95% of the year at a typical site in this land use category.

Source: Martin et al., 2003

In ground water (Exhibit 8-5), EPTC detection frequencies ranged from 0.0% in undeveloped settings to 0.33% in mixed land use (major aquifer) settings, 0.49% in agricultural settings, and 0.72% in urban settings. The 95th percentile concentrations were less than the reporting limit in all settings. The highest concentration, 0.45 µg/L, was found in an agricultural setting (Kolpin and Martin, 2003).

Exhibit 8-5: USGS National Synthesis Summary of NAWQA Monitoring of EPTC in Ambient Ground Water, 1992-2001

Land Use Type	No. of Wells	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,443	0.49%	<RL	<RL	0.45 µg/L
Mixed (Major Aquifer)	2,717	0.33%	<RL	<RL	0.182 µg/L
Undeveloped	67	0.0%	<RL	<RL	<RL
Urban	834	0.72%	<RL	<RL	0.02 µg/L

Notes:

RL = Reporting limit. Reporting limits for EPTC varied, but did not exceed 0.002 µg/L.

The USGS Pesticide National Synthesis considered each well a distinct site in this analysis. Each well was represented by one sample: normally the first one taken, but possibly a later sample if the first sample was not analyzed for the full range of analytes.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Kolpin and Martin, 2003

EPA Summary Analysis of NAWQA Data

Whereas the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination, including EPTC. Detection frequencies were simply computed as the percentage of samples and sites with detections (i.e., with at least one result equal to or greater than the reporting limit). Note that reporting limits were not uniform. Sample detections can be biased by frequent sampling in areas with high (or low) occurrence. Calculating the percentage of sites with detections can reduce this bias. For more details on the data set and the EPA analysis, see Chapter 2.

The results of the EPA analysis are presented in Exhibit 8-6. Overall, EPTC was detected in 10.5% of samples and at 5.7% of sites. EPTC was detected more frequently and at higher concentrations (maximum of 40 µg/L) in surface water.

Exhibit 8-6: EPA Summary Analysis of EPTC Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	14,872	14.4%	1,907	18.9%	0.0004	0.01	0.199	1.5	40
ground water	6,080	0.9%	5,211	0.9%	0.001	0.006	0.17	0.45	0.45
all sites	20,952	10.5%	7,118	5.7%	0.0004	0.01	0.19	1.5	40

[1]RLs (Reporting Limits) for EPTC varied, but did not exceed 0.002 µg/L. For more information, see Chapter 2. Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible.

8.3.3 Drinking Water Occurrence

Nationally representative data on EPTC occurrence in drinking water have been collected by large and small public water systems in accordance with EPA's first Unregulated Contaminant Monitoring Regulation (UCMR 1). For details on UCMR 1, see Chapter 2 and USEPA (2006b).

UCMR 1

UCMR 1 monitoring was conducted primarily between 2001 and 2003, though some results were not collected and reported until as late as 2005. As a List 1 contaminant, EPTC was scheduled to be monitored by all large community water systems (CWSs) and non-transient non-community water systems (NTNCWSs) and a statistically representative sample of small CWSs and NTNCWSs. The data presented in this report reflect UCMR 1 analytical samples submitted and quality-checked under the regulation as of July 2005. EPTC data were collected and submitted by 797 (99.6 percent) of the 800 small systems selected for the small system sample and 3,069 (99.0 percent) of the 3,100 large systems defined as eligible for the UCMR 1 large system census. EPTC data have been analyzed at the level of simple detections (at or above the minimum reporting level, ≥ MRL, or ≥ 1 µg/L), exceedances of the health reference level (> HRL, or > 175 µg/L), and exceedances of one-half the value of the HRL (> ½ HRL, or > 87.5 µg/L).

Results of the analysis are presented in Exhibits 8-7 and 8-8. No detections of EPTC were found in any samples, and thus there were also no exceedances of the HRL or one-half the HRL.

Exhibit 8-7: Summary UCMR 1 Occurrence Statistics for EPTC in Small Systems (Based on Statistically Representative National Sample of Small Systems)

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,251		--
Percent of Samples with Detections	0.00%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	175 µg/L		--
Minimum Reporting Level (MRL)	1 µg/L		--
Maximum Concentration of Detections	< MRL		--
99th Percentile Concentration of Detections	< MRL		--
Median Concentration of Detections	< MRL		--
Total Number of PWSs	797		60,414
Number of GW PWSs	590		56,072
Number of SW PWSs	207		4,342
Total Population	2,760,570		45,414,590
Population of GW PWSs	1,939,815		36,224,336
Population of SW PWSs	820,755		9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%	0
PWSs (GW & SW) > 1/2 HRL	0	0.00%	0
PWSs (GW & SW) > HRL	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	0	0.00%	0
Population Served by PWSs > 1/2 HRL	0	0.00%	0
Population Served by PWSs > HRL	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 8-8: Summary UCMR 1 Occurrence Statistics for EPTC in Large Systems (Based on the Census of Large Systems)

Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	30,384	
Percent of Samples with Detections	0.00%	
99th Percentile Concentration (all samples)	< MRL	
Health Reference Level (HRL)	175 µg/L	
Minimum Reporting Level (MRL)	1 µg/L	
Maximum Concentration of Detections	< MRL	
99th Percentile Concentration of Detections	< MRL	
Median Concentration of Detections	< MRL	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	3,069 1,375 1,694	
Total Population Population of GW PWSs Population of SW PWSs	223,361,341 53,303,000 170,058,341	
Occurrence by System	**Number**	**Percentage**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%
PWSs (GW & SW) > 1/2 HRL	0	0.00%
PWSs (GW & SW) > HRL	0	0.00%
Occurrence by Population Served		
Population Served by PWSs with Detections	0	0.00%
Population Served by PWSs > 1/2 HRL	0	0.00%
Population Served by PWSs > HRL	0	0.00%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Summary Analysis of Combined Large and Small System UCMR 1 Data

None of the 3,866 PWSs sampled (serving a population of 226 million) had detects of EPTC at the MRL of 1 µg/L. Hence, these data indicate that no occurrence and exposure is expected at levels greater than 87.5 µg/L (½ the HRL) and greater than 175 µg/L (the HRL).

Pesticides in Ground Water Database (PGWDB)

The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992). Most of the data are from drinking water wells. Since PGWDB data come from multiple sources, they should be interpreted with caution. Results might be biased high, because areas with suspected contamination are likely to have been sampled more frequently than pristine areas. For more background to the PGWDB, see Chapter 2.

According to the data compiled in the PGWDB, EPTC was found in 2 (0.11 percent) of 1,752 ground water wells that were sampled in 10 States. Both contaminated wells were in Minnesota. The detected concentrations ranged from 0.01 to 0.33 µg/L (USEPA, 1992).

National Pesticide Survey (NPS)

EPA collected samples from approximately 1,300 CWS wells and rural drinking water wells between 1988 and 1990 for the National Pesticide Survey (NPS). The survey was designed to provide a statistically reliable estimate of pesticide occurrence in the nation's drinking water wells. For details about NPS, see Chapter 2.

With a minimum reporting limit of 0.15 µg/L, EPTC was not detected in the survey (USEPA, 1990b).

8.4 Technology Assessment

8.4.1 Analytical Methods

EPA evaluated the availability of analytical methods for all of the unregulated contaminants considered for UCMR 1 (64 FR 50556). Sources for these methods include publications by EPA and by voluntary consensus standard organizations such as the American Society for Testing and Materials (ASTM), the Association of Official Analytical Chemists (AOAC), and the American Public Health Association (APHA).

EPTC is a UCMR 1 List 1 contaminant that can be detected in drinking water by EPA Methods 507 and 525.2. These methods were approved for the monitoring of EPTC in 1999 (64 FR 50556). EPA Method 507 relies on solvent extraction of EPTC and separation by gas chromatography (GC) with a nitrogen-phosphorus detector (NPD), while EPA Method 525.2 relies on liquid-solid extraction and capillary column gas chromatography/ mass spectrometry (GC/MS). A full description of EPA Methods 507 and 525.2 can be found in EPA's *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3* (USEPA, 1995a).

Additional methods approved for EPTC include ASTM Method D5475-93 (ASTM, 1996; 1998) and AOAC 991.07 (AOAC, 1998).

The method detection limit (MDL) and the average recovery for each analytical method used that can be used for the analysis of EPTC in water are included in the method descriptions below.[1]

EPA Method 507

In EPA Method 507 (Revision 2.1), *"Determination of Nitrogen and Phosphorus-Containing Pesticides in Water by Gas Chromatography with a Nitrogen-Phosphorus Detector,"* a sample is extracted with methylene chloride by shaking in a separatory funnel. The methylene chloride extract is separated, dried, and concentrated during a solvent exchange to methyl tert-butyl ether (MTBE). Chromatographic conditions are set to allow for separation and measurement of the analytes in the extract by capillary column GC with a NPD (USEPA, 1995b).

The MDL for EPTC is 0.08 µg/L. The average recovery for EPTC ranges from 83 to 86 percent depending on the method option used (USEPA, 1995b).

EPA Method 525.2

In EPA Method 525.2 (Revision 2.0), *"Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry*," a water sample is passed through a disk or cartridge containing a solid matrix with a chemically bonded C_{18} organic phase. This is known as liquid-solid extraction (LSE). The organic compounds are eluted from the LSE disk or cartridge with small amounts of ethyl acetate and methylene chloride. The analytes are then concentrated by evaporation of some of the solvent. The concentrated extract is analyzed by injecting an aliquot of the extract into the high resolution fused silica capillary column of a GC/MS system. Compounds eluting from the GC column are characterized by comparing their measured mass spectra and retention times against reference mass spectra and retention times (USEPA, 1995c).

[1] The Method Detection Limit (MDL) is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon the precision of a series of replicate measurements of the analyte at low concentrations. The MDL incorporates estimates of the accuracy of the determination. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

The average recovery is the fraction or percent concentration of a target analyte determined relative to the true or expected concentration from a sample containing a known amount of the target analyte. (This can result in apparent recovery values greater than 100 percent.)

The MDL for EPTC in reagent water ranges from 0.056 to 0.12 µg/L, depending on the method option used. The average recovery is reported to range from 97 to 105 percent depending on the method option used (USEPA, 1995c).

8.4.2 Treatment Technologies

Treatment technology status does not influence the determination of whether or not a contaminant should be regulated. However, treatment technologies must be readily available before a contaminant can be regulated with a national primary drinking water regulation (NPDWR). There is no evidence that EPTC is substantially removed by conventional treatments, such as coagulation/flocculation, sedimentation, and inert media filtration. Microbial breakdown has been reported to be a major degradation pathway in soils (Ahrens, 1994 as cited in HSDB, 2004), which suggests the possibility of biological treatment. However, no testing has been done on the biological removal of EPTC from water. Other potential treatment technologies include activated carbon and reverse osmosis.

Granular activated carbon (GAC) treatment removes contaminants via the physical and chemical process of sorption: the contaminants attach to the carbon surface as water passes through the carbon bed. Activated carbon has a large sorption capacity for many water impurities, including synthetic organic chemicals, taste- and odor-causing compounds, and some species of mercury.

Adsorption capacity is typically represented by the Freundlich isotherm constant, with higher Freundlich (K) values indicating greater sorption potential. Activated carbon is considered to be cost-effective for removing a particular contaminant if the Freundlich (K) value of the contaminant is above 200 µg/g $(L/\mu g)^{1/n}$ (Speth *et al.*, 2001). The Freundlich (K) value for EPTC is approximately 79,500 µg/g $(L/\mu g)^{1/n}$, which indicates that GAC is a promising treatment option (Speth *et al.*, 2001).

Reverse osmosis (RO) is similar to other membrane processes, such as ultrafiltration and nanofiltration, in that water passes through a semi-permeable membrane. However, in the case of RO, the membrane is non-porous. RO involves the use of applied hydraulic pressure to oppose the osmotic pressure across the membrane, forcing the water from the concentrated-solution side to the dilute-solution side. The water dissolves into the membrane, diffuses across, then dissolves out into the permeate. Most inorganic and many organic contaminants are rejected by the membrane and will be retained in the concentrate.

USEPA (2000) reports that the carbamate class of pesticides can be removed with 85.7 percent efficiency using a cellulose acetate membrane, 79.6 to 93 percent efficiency using a polyamide membrane, and greater than 92.9 percent efficiency using a thin-film composite membrane. These results indicate that RO is a promising option for removal of EPTC in drinking water.

8.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate EPTC with a national primary drinking water regulation (NPDWR). Because EPTC does not appear to occur at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. While EPTC has been found in ambient waters, it was detected only at levels less than the HRL (as well as ½ the HRL) and it was not found in the UCMR 1 survey of public water supplies.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

8.6 References

Ahrens, W.H. 1994. *Herbicide Handbook of the Weed Science Society of America*. 7th Edition, Weed Science Society of America, Champaign, IL. (As cited in HSDB, 2004).

American Society for Testing and Materials (ASTM). 1996. *Annual Book of ASTM Standards.* Volume 11.02. West Conshohocken, PA: American Society for Testing and Materials.

ASTM. 1998. *Annual Book of ASTM Standards.* Volume 11.02. West Conshohocken, PA: American Society for Testing and Materials.

Association of Official Analytical Chemist (AOAC) International. 1998. *Official Methods of Analysis of AOAC International.* 16th ed. 4th Revision. Volume I. Baltimore, MD: AOAC International.

Hansch, C., A.J. Leo, and D. Hoekman. 1995. *Exploring QSAR – Hydrophobic, Electronic, and Steric Constants*. Washington, DC: American Chemical Society. (As cited in HSDB, 2004).

Hazardous Substances Data Bank (HSDB). 2004. Search for Eptam. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB, accessed November 1, 2004.

Kolpin, D.W. and J.D. Martin. 2003. Pesticides in Ground Water: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestgw/Pest-GW_2001_Text.html, accessed August 24, 2004. Link to document from: http://ca.water.usgs.gov/pnsp/.

Martin, J.D., C.G. Crawford, and S.J. Larson. 2003. Pesticides in Streams: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestsw/Pest-SW_2001_Text.html, accessed August 24, 2004. Link to document from: http://ca.water.usgs.gov/pnsp/.

National Center for Food and Agricultural Policy (NCFAP). 2004. National Pesticide Use Database. Available on the Internet at: http://www.ncfap.org/database/default.htm.

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

Thelin, G.P. and L.P. Gianessi. 2000. *Method for Estimating Pesticide Use for County Areas of the Conterminous United States.* U.S. Geological Survey Open-File Report 00-250, 62 p. Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/ofr00250/ofr00250.pdf, accessed August 30, 2004.

Mackenzie, K. 1986. Two-generation Reproduction Study with EPTC in Rats: Report: Study No. 6100-108. Unpublished study prepared by Hazleton Laboratories America, Inc. 1192 p. (As cited in USEPA, 1999a).

Tomlin, C.D.S. (ed.) 1997. *The Pesticide Manual – World Compendium*. 11th edition. Surrey, England: British Crop Protection Council. (As cited in HSDB, 2004).

United States Department of Agriculture (USDA). 2000. ARS Pesticide Properties Database on EPTC (759-94-4). Available from the Database Query page at: http://www.arsusda.gov/acsl/services/ppdb/ppdb3.html, as of Dec 15, 2004. (As cited in HSDB, 2004).

United States Environmental Protection Agency (USEPA). 1990a. United States Environmental Protection Agency. Integrated Risk Information System (IRIS) for EPTC. Cincinnati, OH. Available at: http://www.epa.gov/iriswebp/iris/subst/0237.htm

USEPA. 1990b. *National Pesticide Survey: Survey Analytes.* EPA Report 570-9-90-NPS2. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS2.]

USEPA. 1992. *Pesticides in Ground Water Database: A Compilation of Monitoring Studies, 1971-1991. National Summary.* Office of Prevention, Pesticides and Toxic Substances. EPA Report 734-12-92-001. Available on the Internet at: http://nepis.epa.gov/pubtitleOPPTS.htm. [Search for document number 7341292001.]

USEPA. 1995a. *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. Office of Research and Development, EPA 600-R-95-131.

USEPA. 1995b. Method 507 - Determination of Nitrogen and Phosphorus-Containing Pesticides in Water by Gas Chromatography with a Nitrogen-Phosphorus Detector. Revision 2.1. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. Office of Research and Development, EPA 600-R-95-131.

USEPA. 1995c. Method 525.2 - Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry.

Revision 2.0. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. Office of Research and Development, EPA 600-R-95-131.

USEPA. 1999a. *Reregistration Eligibility Decision (RED): EPTC*. EPA Report 738-R-99-006. Washington, DC: Office of Prevention, Pesticides and Toxic Substances. Available on the Internet at: http://www.epa.gov/oppsrrd1/REDs/0064red.pdf.

USEPA. 1999b. Guidelines for carcinogen risk assessment. Review Draft. Environmental Protection Agency Risk Assessment Forum. NCEA-F-0644. July 1999.

USEPA. 2000. *Summary of Pesticide Removal/Transformation Efficiencies from Various Drinking Water Treatment Processes*. Report prepared for the Committee to Advise on Reassessment And Transition (CARAT). Available on the Internet at: http://www.epa.gov/oppfead1/carat/2000/oct/dw4.pdf.

USEPA. 2002. *Pesticides Industry Sales and Usage. 1998 and 1999 Market Estimates.* EPA 733-R-02-001. Office of Prevention, Pesticides, and Toxic Substances. Available on the Internet at: http://www.epa.gov/oppbead1/pestsales/99pestsales/market_estimates1999.pdf. Accessed August 30, 2004.

USEPA. 2006a. TRI Explorer: Trends. Search for ethyl dipropylthiocarbamate. Available on the Internet at: http://www.epa.gov/triexplorer/trends.htm. Last modified June 8, 2005, accessed February 8, 2006.

USEPA. 2006b. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

United State Geological Survey (USGS). 2004. 1997 Pesticide Use Maps. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pesticide_use_maps_1997/, accessed August 17, 2004, last updated August 14, 2003.

Windholz, M., S. Budavari, R.F. Blumetti, and E.S. Otterbein (eds.) 1983. *The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals.* 10th ed. Rahway, NJ: Merck and Co., Inc.

Yalkowsky, S.H. and R.M. Dannenfelser. 1992. The AQUASOL Database of Aqueous Solubility. Version 5. Tucson, AZ: University of Arizona, College of Pharmacy. (As cited in HSDB, 2004).

Chapter 9:
Fonofos

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

Fonofos, an organophosphate, is a soil insecticide used until recently to control pests such as corn rootworms, cutworms, symphylans (i.e., garden centipedes), and wireworms. Primarily used on corn crops, fonofos was also used on other crops such as asparagus, beans, beets, corn, onions, peppers, tomatoes, cole crops, sweet potatoes, peanuts, peas, peppermint, plantains, sorghum, soybeans, spearmint, strawberries, sugarcane, sugar beets, white (Irish) potatoes, and tobacco.

Fonofos was scheduled for a reregistration decision in 1999. However, before the review was completed, the registrant requested voluntary cancellation. The cancellation was announced in the *Federal Register* on May 6, 1998 (63 FR 25033), with an effective date of November 2, 1998, plus a one-year grace period to permit the exhaustion of existing stocks.

Fonofos is moderately persistent in soil and its persistence depends on soil type, organic matter, rainfall, and sunlight. Since fonofos adsorbs moderately well to soil, it is not readily leached or transported to ground water but it can be transported to surface waters in runoff. Fonofos is rapidly degraded by soil microorganisms. Fonofos tends to volatilize from wet soil and water surfaces, but the process is slowed by adsorption to organic material in soil, suspended solids, and sediment.

Fonofos (like many organophosphates) is toxic to humans and animals. Case reports and acute oral toxicity studies in animals indicate that oral exposure to fonofos induces clinical signs of toxicity that are typical of cholinesterase inhibitors. Chronic exposure studies also indicated that oral administration of fonofos inhibits cholinesterase. Cholinesterase inhibition is one of the critical effects associated with the reference dose (RfD), which was verified by EPA at 0.002 mg/kg/day. EPA derived the RfD using an no-observed-adverse-effect level (NOAEL) of 0.2 mg/kg/day and a 100-fold uncertainty factor to account for inter- and intraspecies differences.

Fonofos is classified as an unlikely human carcinogen (Group E) because available long-term feeding studies in rats and mice show no evidence of carcinogenicity. Fonofos does not appear to be mutagenic.

The Agency believes that the current RfD is adequately protective of children. The current fonofos RfD of 0.002 mg/kg/day is 1000-fold lower than the NOAEL observed in rat developmental studies. Using the RfD of 0.002 mg/kg/day for fonofos and a 20 percent screening relative source contribution, the Agency derived a health reference level (HRL) of 0.014 mg/L and rounded to 0.01 mg/L (or 10 µg/L).

National Center for Food and Agricultural Policy (NCFAP) data indicate that fonofos use declined significantly during the 1990s. According to NCFAP, approximately 3.2 million pounds of fonofos were applied annually around 1992 and approximately 0.4 million pounds were applied annually around 1997. Fonofos use was cancelled in 1998.

Data on the ambient occurrence of fonofos are available from the first monitoring cycle (1992-2001) of the United States Geological Survey's (USGS's) National Ambient Water

Quality Assessment (NAWQA) program. While the USGS detected fonofos in both surface and ground waters, in no land use setting did the 95th percentile concentration of fonofos exceed 0.003 µg/L (the reporting limit). The maximum surface water concentration, 1.20 µg/L (from an agricultural setting), and the maximum ground water concentration, 0.009 µg/L (also from an agricultural setting), are both less than the fonofos HRL and ½ the HRL.

To estimate fonofos occurrence in drinking water, EPA included it as an analyte in the first Unregulated Contaminant Monitoring Regulation (UCMR 1) List 2 Screening Survey. None of the 2,306 samples from the 295 public water systems (PWSs) sampled (serving a total population of 41 million) had fonofos detections at or above the minimum reporting level (MRL) of 0.5 µg/L. These results suggest that no occurrence and exposure is expected at levels greater than the HRL (10 µg/L) or even ½ the HRL (5 µg/L).

The Agency has made a preliminary determination not to regulate fonofos with a national primary drinking water regulation (NPDWR). Because fonofos does not appear to occur at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

a.i.	Active Ingredient
AOAC	Association of Official Analytical Chemists
APHA	American Public Health Association
ASTM	American Society for Testing and Materials
CAS	Chemical Abstracts Service
CCL 2	Second Contaminant Candidate List
GAC	Granular Activated Carbon
GC/MS	Gas Chromatography with Mass Spectrometry
HRL	Health Reference Level
MDL	Method Detection Limit
MRL	Minimum Reporting Level
NAWQA	National Water Quality Assessment
NCFAP	National Center for Food and Agricultural Policy
NOAEL	No Observed Adverse Effect Level
NPDWR	National Primary Drinking Water Regulation
PGWDB	Pesticides in Ground Water Database
PWS	Public Water System
RfD	Reference Dose
RL	Reporting Limit
RO	Reverse Osmosis
SDVB	Polystyrenedivinylbenzene
SPE	Solid Phase Extraction
UCMR 1	First Unregulated Contaminant Monitoring Regulation
USGS	United States Geological Survey

9 Fonofos

9.1 Definition

Fonofos is a highly toxic organophosphate insecticide. The Chemical Abstracts Service (CAS) chemical name for fonofos is O-ethyl S-phenyl ethylphosphonodithioate, and its registry number is 944-22-9. Trade name synonyms include Difonate, Dyfonate, Dyphonate, Capfos, Cudgel, and Stauffer N 2790 (Extoxnet, 1993). Two chiral forms of fonofos exist, of which the (R)-isomer is more toxic to mice and insects than the (S)-isomer (Tomlin, 2002 as cited in HSDB, 2004).

9.1.1 Properties and Sources

At room temperature, fonofos is a clear-to-yellow liquid with a distinct mercaptan (sulfur) odor. As a synthetic compound, it is not found naturally in the environment. Fonofos is applied to soil to control insects around crops (predominantly corn). It is relatively insoluble in water, but miscible in most common organic solvents. Fonofos is available in a variety of formulations, including granular, microgranular, emusifiable concentrate, and suspension concentrate forms (Extoxnet, 1993). Some additional physical and chemical properties of fonofos are listed in Exhibit 9-1.

Exhibit 9-1: Physical and Chemical Properties of Fonofos

Identification	
CAS number	944-22-9
Molecular Formula	$C_{10}H_{15}OPS_2$
Physical and Chemical Properties	
Boiling Point	130 °C at 0.1 mm Hg [1]
Melting Point	< 25 ° C [1]
Molecular Weight	246.32 g/mol [1]
Log K_{oc}	870 [2]
Log K_{ow}	3.94 [3]
Water Solubility	15.7 mg/L at 20 °C [4]
Vapor Pressure	0.000338 mm Hg at 25 ° C [5]
Henry's Law Constant	7.0×10^{-6} atm-m^3/mole [6] 2.1×10^{-4} (dimensionless), predicted [7] 2.6×10^{-4} (dimensionless), from literature [7]
Freundlich Isotherm Constant (K)	251,000 (µg/g)(L/µg)$^{1/n}$ [7]

[1] *Windholz et al., 1983*

[2] *Wauchope at al., 1992 (as cited in Extoxnet, 1993)*

[3] *Hansch et al., 1995 (as cited in HSDB, 2004)*

[4] *Yalkowsky & He, 2003 (as cited in HSDB, 2004)*

[5] *USDA, 2003 (as cited in HSDB, 2004)*

[6] *HSDB, 2004*

[7] *Speth et al., 2001*

9.1.2 Environmental Fate and Behavior

Fonofos is moderately persistent in soil and its persistence depends on soil type, organic matter, rainfall, and sunlight. Since fonofos adsorbs moderately well to soil, it is not readily leached or transported to ground water but it can be transported to surface waters in runoff. Fonofos is rapidly degraded by soil microorganisms (Extoxnet, 1993). Fonofos tends to volatilize from wet soil and water surfaces, but the process is slowed by adsorption to organic material in soil, suspended solids, and sediment (HSDB, 2004).

According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere, fonofos will exist in both the vapor and particulate phases in the ambient atmosphere (Bidleman, 1988 as cited in HSDB, 2004). In a laboratory volatility study, approximately 35 percent of the fonofos that was applied to soil volatilized after 24 hours (USEPA, 1999).

9.2 Health Effects

Fonofos (like many organophosphates) is toxic to humans and animals. Case reports and acute oral toxicity studies in animals indicate that oral exposure to fonofos induces clinical signs of toxicity that are typical of cholinesterase inhibitors. In humans, accidental exposures produced symptoms of acute intoxication, nausea, vomiting, salivation, sweating, muscle twitches, decreased blood pressure and pulse rate, pinpoint pupils, profuse salivary and bronchial secretions, cardiorespiratory arrest, and even death in one exposed individual (Hayes, 1982 as cited in USEPA 1988; Pena Gonzalez *et al.*, 1996).

In animals, clinical signs of exposure included tremors, salivation, diarrhea, and labored breathing (USEPA, 1996). Chronic exposure studies also indicated that oral administration of fonofos inhibits cholinesterase (Banerjee *et al.*, 1968; Cockrell *et al.*, 1966; both as cited in USEPA, 1988; Hodge, 1995; Horner, 1993; Pavkov and Taylor, 1988; Woodard *et al.*, 1969 both as cited in USEPA, 1996; Miller, 1987 as cited in USEPA 1996; Miller *et al.*, 1979). Cholinesterase inhibition is one of the critical effects associated with the reference dose (RfD), which was verified by EPA (1991) at 0.002 mg/kg/day. EPA derived the RfD value of 0.002 mg/kg/day using a no-observed-adverse-effect level (NOAEL) of 0.2 mg/kg/day (Hodge, 1995 as cited in USEPA, 1996) and a 100-fold uncertainty factor to account for inter- and intraspecies differences.

Fonofos is classified as an unlikely human carcinogen (Group E) because there is no evidence of carcinogenic potential in the available long-term feeding studies in rats and mice (Banerjee *et al.* 1968 as cited in USEPA, 1988; Pavkov and Taylor, 1988, Sprague and Zwicker, 1987 both as cited in USEPA, 1996). In addition, fonofos does not appear to be mutagenic (USEPA, 1996).

EPA evaluated whether health information is available regarding the potential effects on children and other sensitive populations. In the available developmental studies with rabbits (Sauerhoff, 1987 as cited in USEPA, 1996) and mice (Minor *et al.*, 1982 as cited in USEPA, 1988; Pulsford, 1991 as cited in USEPA, 1996), no developmental effects were observed at oral doses as high as 1.5 mg/kg/day in the rabbit (highest dose tested) nor in mice at doses as high as 2.0 mg/kg/day (Minor *et al.*, 1982 as cited in USEPA, 1988; Pulsford, 1991 as cited in USEPA, 1996). However, in mice, effects were noted at higher dose levels. These effects include an increase in the incidence of variant sternebrae ossifications (at 6 mg/kg/day or greater) and a slight dilation of the fourth brain ventricle in offspring (at 4 mg/kg/day or greater). No developmental neurotoxicity study with fonofos is available for further assessment of this endpoint. In a three-generation reproduction study in rats (Woodard *et al.*, 1968 as cited in USEPA, 1996), no treatment-related adverse effects were observed at the two dose levels used in this study, 0.5 and 1.58 mg/kg/day.

The Agency believes that the current RfD is adequately protective of children. The current fonofos RfD of 0.002 mg/kg/day is 1000-fold lower than the NOAEL observed in the Woodard *et al.* (1968 as cited in USEPA, 1996) developmental studies.

Using the RfD of 0.002 mg/kg/day for fonofos and a 20 percent screening relative source contribution, the Agency derived a health reference level (HRL) of 0.014 mg/L and rounded to 0.01 mg/L (or 10 µg/L).

9.3 Occurrence and Exposure

9.3.1 Use and Environmental Release

Fonofos, a highly toxic liquid organophosphate insecticide, was initially marketed in 1967 by Stauffer Chemical Company, and most recently licensed to Zeneca Ag Products. Fonofos was used primarily on corn crops, but was also applied to others, including asparagus, beans, beets, corn, onions, peppers, tomatoes, cole crops, sweet potatoes, peanuts, peas, peppermint, plantains, sorghum, soybeans, spearmint, strawberries, sugarcane, sugar beets, white (Irish) potatoes, and tobacco. Applied at rates between 1 and 4 pounds per acre, fonofos was used to control insects such as corn rootworms, cutworms, symphylans (garden centipedes), and wireworms (USEPA, 1999).

In March 1984, EPA issued a Registration Standard for fonofos. Although fonofos was scheduled for a reregistration decision in 1999, the registrants requested voluntary cancellation before the review. Cancellation of the pesticide was announced in the Federal Register on May 8, 1998 (63 FR 25033), with an effective date of November 2, 1998, plus a one-year grace period to permit the exhaustion of existing stocks (USEPA, 1999).

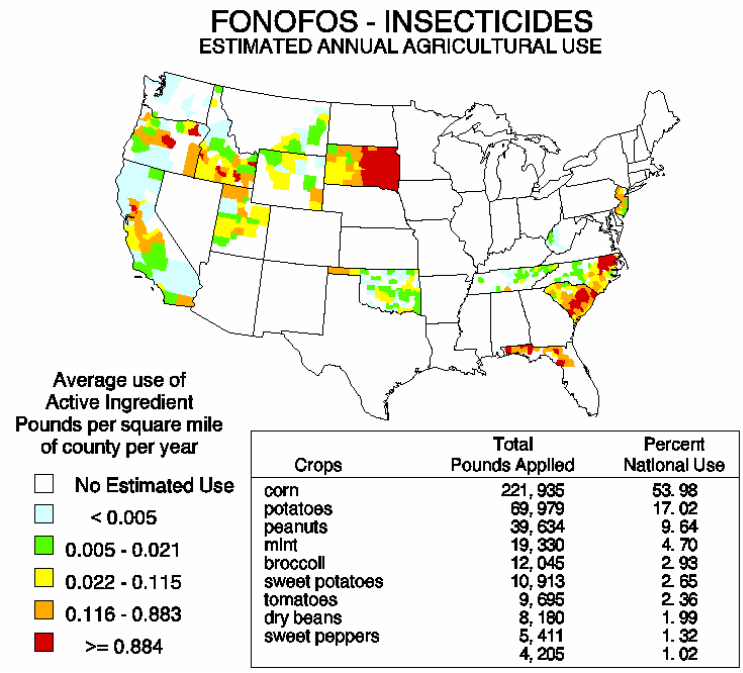
The National Center for Food and Agricultural Policy (NCFAP) estimates of national fonofos use indicate a significant decline during the 1990s (NCFAP, 2004). According to NCFAP, approximately 3.2 million pounds of active ingredient (a.i.) were applied annually to 24 types of crops on 2.6 million acres around 1992, and approximately 0.4 million pounds a.i. were applied annually to 19 types of crops on 0.3 million acres around 1997. NCFAP estimates are based on State-level commercial agriculture usage patterns for the periods 1990-1993 and 1995-1998, and State-level crop acreage for 1992 and 1997. For more information on NCFAP pesticide use estimates, see Chapter 2.

The United States Geological Survey (USGS) combined data collected by NCFAP with data from the Census of Agriculture to estimate that 2.7 million pounds of fonofos a.i. per year were used on approximately 2.4 million agricultural acres in the early 1990s (Thelin and Gianessi, 2000). While USGS has not published national estimates for 1997, an estimate of approximately 0.4 million pounds a.i. can be inferred from the "total pounds applied" and "percent national use" data in the 1997 geographical distribution map (Exhibit 9-2).

Exhibit 9-2 shows the estimated geographic distribution and intensity of typical annual fonofos use in the United States in the late 1990s. A breakdown of use by crop is also included. The map was created by USGS using State-level data sets on pesticide use rates from 1995-1998 compiled by NCFAP, combined with county-level data on harvested crop acreage obtained from the 1997 Census of Agriculture (USGS, 2004). Due to the nature of the data sources, non-agricultural uses are not reflected on the map and variations in use at the county-level are also not well represented (Thelin and Gianessi, 2000). For background on the USGS pesticide use

maps, see Chapter 2. The map suggests that around 1997, fonofos was used in a geographically dispersed minority of States, most intensely in South Dakota.

Exhibit 9-2: Estimated Annual Agricultural Use of Fonofos, c. 1997



Crops	Total Pounds Applied	Percent National Use
corn	221,935	53.98
potatoes	69,979	17.02
peanuts	39,634	9.64
mint	19,330	4.70
broccoli	12,045	2.93
sweet potatoes	10,913	2.65
tomatoes	9,695	2.36
dry beans	8,180	1.99
sweet peppers	5,411	1.32
	4,205	1.02

Source: USGS, 2004

9.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are the source of most drinking water. Data on the occurrence of fonofos in ambient surface and ground water are available from the National Water Quality Assessment (NAWQA) program of the USGS. For details on this program, see the discussion in Chapter 2. NAWQA data have been analyzed independently by USGS and EPA.

NAWQA National Pesticide Synthesis

Under the NAWQA program, USGS monitored fonofos between 1992 and 2001 in representative watersheds and aquifers across the country. Reporting limits varied but did not exceed 0.003 µg/L.

In surface water (Exhibit 9-3), fonofos was detected at frequencies ranging from 0.0% of samples in undeveloped land settings to 0.92% in urban land use settings, 1.20% in mixed land use settings, and 3.05% in agricultural land use settings. The 95th percentile concentrations in all land use settings were below the reporting limit. The highest concentration, 1.20 µg/L, occurred in an agricultural land use setting (Martin *et al.*, 2003).

Exhibit 9-3: USGS National Synthesis Summary of NAWQA Monitoring of Fonofos in Ambient Surface Water, 1992-2001

Land Use Type	No. of Samples (and No. of Sites)	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,889 (78)	3.05%	<RL	<RL	1.20 µg/L
Mixed	1,020 (47)	1.20%	<RL	<RL	0.014 µg/L
Undeveloped	60 (4)	0.00%	<RL	<RL	<RL
Urban	900 (33)	0.92%	<RL	<RL	0.084 µg/L

Notes:

RL = Reporting limit. Reporting limits for fonofos varied, but did not exceed 0.003 µg/L.

The USGS Pesticide National Synthesis used one year of data, generally the year with the most sampling results, to represent each site in this analysis. The sampling results were time-weighted to eliminate bias from more frequent sampling at certain times of year. Detection Frequencies and Percentile Concentrations can be interpreted as representing annual occurrence. For instance, the detection frequency can be thought of as the percent of the year in which detections are found at a typical site in this land use category, and the 95th percentile concentration can be thought of as a concentration that is not exceeded for 95% of the year at a typical site in this land use category.

Source: Martin et al., 2003

In ground water, fonofos detection frequencies ranged from 0.0% of samples in urban and undeveloped settings to 0.07% in agricultural and mixed land use (major aquifer) settings (Exhibit 9-4). The 95th percentile concentrations were less than the reporting limit in all settings. The highest concentration, 0.009 µg/L, occurred in an agricultural setting (Kolpin and Martin, 2003).

Exhibit 9-4: USGS National Synthesis Summary of NAWQA Monitoring of Fonofos in Ambient Ground Water, 1992-2001

Land Use Type	No. of Wells	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,443	0.07%	<RL	<RL	0.009 µg/L
Mixed (Major Aquifer)	2,717	0.07%	<RL	<RL	0.003 µg/L
Undeveloped	67	0.0%	<RL	<RL	<RL
Urban	835	0.0%	<RL	<RL	<RL

Notes:

RL = Reporting limit. Reporting limits for fonofos varied, but did not exceed 0.003 µg/L.

The USGS Pesticide National Synthesis considered each well a distinct site in this analysis. Each well was represented by one sample: normally the first one taken, but possibly a later sample if the first sample was not analyzed for the full range of analytes.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Kolpin and Martin, 2003

EPA Summary Analysis of NAWQA Data

Whereas the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination, including fonofos. Detection frequencies were simply computed as the percentage of samples and sites with detections (i.e., with at least one result equal to or greater than the reporting limit). Note that reporting limits were not uniform. Sample detections can be biased by frequent sampling in areas with high (or low) occurrence. Calculating the percentage of sites with detections can reduce this bias. For more details on the data set and the EPA analysis, see Chapter 2.

The results of the EPA analysis are presented in Exhibit 9-5. Overall, fonofos was detected in 2.20% of samples and at 1.34% of sites. Fonofos was detected more frequently and at higher concentrations (maximum of 1.2 µg/L) in surface water.

Exhibit 9-5: EPA Summary Analysis of Fonofos Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	14,880	3.08%	1,907	4.82%	0.0005	0.007	0.073	0.21	1.2
ground water	6,078	0.05%	5,209	0.06%	0.002	0.003	0.009	0.009	0.009
all sites	20,958	2.20%	7,116	1.34%	0.0005	0.007	0.07	0.21	1.2

[1]*RLs (reporting limits) for fonofos varied but did not exceed 0.003 µg/L. See Chapter 2 for more information. Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible.*

9.3.3 Drinking Water Occurrence

Nationally representative data on fonofos occurrence in drinking water have been collected by large and small public water systems in accordance with EPA's first Unregulated Contaminant Monitoring Regulation (UCMR 1). For a detailed description of UCMR 1, see Chapter 2 and USEPA (2006).

UCMR 1

UCMR 1 monitoring was conducted primarily between 2001 and 2003. As a List 2 contaminant, fonofos was scheduled to be monitored by 300 public water systems, including both large and small systems. The data presented in this report reflect UCMR 1 analytical samples submitted and quality-checked under the regulation as of July 2005. Fonofos data were collected and submitted by 178 (98.9 percent) of the 180 small systems selected for the small system sample and 117 (97.5 percent) of the 120 large systems selected for the large system sample. These included two systems in South Dakota, twelve systems in North Carolina, and four systems in South Carolina (States where fonofos use is particularly intensive). The data have been analyzed at the level of simple detections (at or above the minimum reporting level, ≥ MRL, or ≥ 0.5 µg/L), exceedances of the health reference level (> HRL, or > 10 µg/L), and exceedances of one-half the value of the HRL (> ½ HRL, or > 5 µg/L).

Results of the analysis are presented in Exhibits 9-6 and 9-7. No detections of fonofos were found in any samples, and thus there were also no exceedances of the HRL or one-half the HRL.

Exhibit 9-6: Summary UCMR 1 Occurrence Statistics for Fonofos in Small Systems

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	643		--
Percent of Samples with Detections	0.00%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	10 µg/L		--
Minimum Reporting Level (MRL)	0.5 µg/L		--
Maximum Concentration of Detections	< MRL		--
99th Percentile Concentration of Detections	< MRL		--
Median Concentration of Detections	< MRL		--
Total Number of PWSs Number of GW PWSs Number of SW PWSs	178 114 64		60,414 56,072 4,342
Total Population Population of GW PWSs Population of SW PWSs	508,136 275,185 232,951		45,414,590 36,224,336 9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%	0
PWSs (GW & SW) > 1/2 HRL	0	0.00%	0
PWSs (GW & SW) > HRL	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	0	0.00%	0
Population Served by PWSs > 1/2 HRL	0	0.00%	0
Population Served by PWSs > HRL	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 9-7: Summary UCMR 1 Occurrence Statistics for Fonofos in Large Systems

Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	1,663	
Percent of Samples with Detections	0.00%	
99th Percentile Concentration (all samples)	< MRL	
Health Reference Level (HRL)	10 µg/L	
Minimum Reporting Level (MRL)	0.5 µg/L	
Maximum Concentration of Detections	< MRL	
99th Percentile Concentration of Detections	< MRL	
Median Concentration of Detections	< MRL	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	117 50 67	
Total Population Population of GW PWSs Population of SW PWSs	40,259,344 8,000,122 32,259,222	
Occurrence by System	**Number**	**Percentage**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%
PWSs (GW & SW) > 1/2 HRL	0	0.00%
PWSs (GW & SW) > HRL	0	0.00%
Occurrence by Population Served		
Population Served by PWSs with Detections	0	0.00%
Population Served by PWSs > 1/2 HRL	0	0.00%
Population Served by PWSs > HRL	0	0.00%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Summary Analysis of Combined Large and Small System UCMR 1 Data

None of the 2,306 samples from the 295 public water systems (PWSs) sampled (serving a population of 41 million) contained detects for fonofos at the MRL of 0.5 µg/L. Hence, these data indicate that no occurrence and exposure is expected at levels greater than 5 µg/L (½ the HRL) and greater than 10 µg/L (the HRL).

Pesticides in Ground Water Database (PGWDB)

The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992). Most of the data are from drinking water wells. Since PGWDB data come from multiple sources, they should be interpreted with caution. Results might be biased high, because areas with suspected contamination are likely to have been sampled more frequently than pristine areas. For more information on the PGWDB, see Chapter 2.

According to the data compiled in the PGWDB, fonofos was detected in 18 (0.4 percent) of 4,446 wells sampled. The detections were found in 5 out of 13 States where fonofos was investigated. Concentrations ranged from 0.11 to 0.90 µg/L in Iowa, from 0.007 to 0.05 µg/L in Oregon, and from 0.007 to 0.06 µg/L in South Dakota; one Montana well had a concentration of 0.43 µg/L and one Maine well had a concentration of 0.05 µg/L. These detections were all well below the HRL of 10 µg/L (USEPA, 1992).

9.4 Technology Assessment

9.4.1 Analytical Methods

EPA evaluated the availability of analytical methods for all of the unregulated contaminants considered for UCMR 1 (64 FR 50556; September 17, 1999). Sources for these methods include publications by EPA and voluntary consensus standard organizations, such as the American Society for Testing and Materials (ASTM), the Association of Official Analytical Chemists (AOAC), and the American Public Health Association (APHA).

Fonofos is a UCMR 1 List 2 contaminant that can be detected in drinking water using EPA Method 526. This method was approved in the UCMR 1 List 2 Rule (66 FR 2273; January 11, 2001) for monitoring fonofos. EPA Method 526 relies on solid phase extraction (SPE) followed by capillary column gas chromatography coupled with mass spectrometry (GC/MS). A full description of EPA Method 526 can be found in EPA's *Methods for the Determination of Organic and Inorganic Compounds in Drinking Water, Volume 1* (USEPA, 2000a). A brief summary of the method is provided below.

EPA Method 526

For EPA Method 526 (Revision 1.0), *"Determination of Selected Semivolatile Organic Compounds in Drinking Water by Solid Phase Extraction and Capillary Column Gas Chromatography/Mass Spectrometry (GC/MS),"* target analytes are extracted from a water sample by passing the water through a SPE disk or cartridge containing polystyrenedivinylbenzene (SDVB). The extract is then dried, concentrated and diluted by the addition of internal standards. An aliquot of the extract is injected into a gas chromatograph with a high resolution fused silica capillary column to separate the components. The analytes are transferred from the capillary column to the mass spectrometer and subsequently identified. Mass spectrometry is advantageous as a detection method since it reports few false positive results compared to conventional detection methods (USEPA, 2000b).

The MDL for fonofos demonstrated by Method 526 ranges from 0.022 to 0.06 µg/L depending upon the extraction media used (USEPA, 2000b). The average recovery for fonofos using Method 526 ranges from 89 to 109 percent, depending on the method option used (USEPA, 2000b).[1]

9.4.2 Treatment Technologies

Treatment technology status does not influence the determination of whether or not a contaminant should be regulated. However, treatment technologies must be readily available before a contaminant can be regulated with a national primary drinking water regulation (NPDWR). There is no evidence that fonofos is substantially removed by conventional treatments, such as coagulation/flocculation, sedimentation, and inert media filtration. Potential treatment technologies include activated carbon, reverse osmosis, and advanced oxidation.

Granular activated carbon (GAC) treatment removes contaminants via the physical and chemical process of sorption: the contaminants attach to the carbon surface as water passes through the carbon bed. Activated carbon has a large sorption capacity for many water impurities, including synthetic organic chemicals, taste- and odor-causing compounds, and some species of mercury.

Adsorption capacity is typically represented by the Freundlich isotherm constant, with higher Freundlich (K) values indicating greater sorption potential. Activated carbon is considered to be cost-effective for removing a particular contaminant if the Freundlich (K) value

[1] The Method Detection Limit (MDL) is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon the precision of a series of replicate measurements of the analyte at low concentrations. The MDL incorporates estimates of the accuracy of the determination. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

The average recovery is the fraction or percent concentration of a target analyte determined relative to the true or expected concentration from a sample containing a known amount of the target analyte. (This can result in apparent recovery values greater than 100 percent.)

of the contaminant is above 200 µg/g $(L/\mu g)^{1/n}$ (Speth *et al.*, 2001). Speth *et al.* (2001) report that the Freundlich (K) value for fonofos is 251,000 µg/g $(L/\mu g)^{1/n}$, which indicates that GAC is a promising treatment option.

Reverse osmosis (RO) is similar to other membrane processes, such as ultrafiltration and nanofiltration, in that water passes through a semi-permeable membrane. However, in the case of RO, the membrane is non-porous. RO involves the use of applied hydraulic pressure to oppose the osmotic pressure across the membrane, forcing the water from the concentrated-solution side to the dilute-solution side. The water dissolves into the membrane, diffuses across, then dissolves out into the permeate. Most inorganic and many organic contaminants are rejected by the membrane and will be retained in the concentrate.

USEPA (2000c) report that the organophosphate class of pesticides can be removed with 97.8 to 99 percent efficiency using a cellulose acetate membrane and 98.5 to 100 percent efficiency using a thin-film composite membrane. These results indicate that RO is a promising option for removal of fonofos in drinking water.

9.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate fonofos with a national primary drinking water regulation (NPDWR). Because fonofos does not appear to occur at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. While fonofos has been found in ambient waters, it was detected only at levels less than the HRL (as well as ½ the HRL) and it was not found in UCMR 1 Screening Survey of public water supplies. Fonofos was voluntarily cancelled in 1998 and the Agency expects any remaining stocks and releases into the environment to decline. In addition, since fonofos tends to bind strongly to soil, any releases to the environment are not likely to contaminant source waters.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

9.6 References

Banerjee, B.M., D. Howard, and M.W. Woodard. 1968. *Dyfonate (N-2790) safety evaluation by dietary administration to rats for 105 weeks.* (As cited in USEPA, 1988).

Bidleman, T.F. 1988. Atmospheric processes. *Environmental Science and Technology*. 22:361-367. (As cited in HSDB, 2004).

Cockrell, K.O., M.W. Woodard, and G. Woodard. 1966. *N-2790 Safety evaluation by repeated oral administration to dogs for 14 weeks and to rats for 13 weeks.* (As cited in USEPA, 1988).

Extoxnet. 1993. "Extension Toxicology Network Pesticide Information Profiles–Fonofos." Available on the Internet at: http://pmep.cce.cornell.edu/profiles/extoxnet/dienochlor-glyphosate/fonofos-ext.html. Accessed March 10, 2006.

Hansch, C., A.J. Leo, and D. Hoekman. 1995. *Exploring QSAR – Hydrophobic, Electronic, and Steric Constants*. Washington, DC: American Chemical Society. (As cited in HSDB, 2004).

Hayes, W.J. 1982. Pesticides Studied in Man. Baltimore, MD: Williams and Wilkins. P. 413. (As cited in USEPA, 1988).

Hazardous Substances Data Bank (HSDB). 2004. Search for Fonofos. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed November 1, 2004.

Hodge, M. 1995. *Fonofos: 1-Year Old Toxicity Study in Dogs*. Report Number CTL/P/4499, Study Number PDO944. Zeneca Central Toxicology Lab. (As cited in USEPA, 1996).

Horner, J. 1993. *Fonofos: Subchronic Neurotoxicity Study in Rats*. Lab Project Number: CTL/P/3879, PRO889. Zeneca Central Toxicology Lab. (As cited in USEPA, 1996).

Kolpin, D.W. and J.D. Martin. 2003. "Pesticides in Ground Water: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestgw/Pest-GW_2001_Text.html. Accessed August 24, 2004.

Martin, J.D., C.G. Crawford, and S.J. Larson. 2003. "Pesticides in Streams: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestsw/Pest-SW_2001_Text.html. Accessed August 24, 2004.

Miller, J.L., L. Sandvik, G.L. Sprague, A.A. Bickford, and T.R. Castles. 1979. Evaluation of delayed neurotoxic potential of chronically administered Dyfonate in adult hens. Abstract presented in *Toxicology and Applied Pharmacology*. 48(Part 2):A197.

Miller, J. 1987. *Neurotoxicity of 90-Day Oral Administration of Technical Dyfonate to Adult Hens*. T-6237: Final Report. Stauffer Chemical Co. p. 59. (As cited in USEPA, 1996).

Minor, J., J. Downs, G. Zwicker, *et al.* 1982. *A teratology study in CD-1 mice with Dyfonate technical.* T-10192. Final report. Stauffer Chemical Company. (As cited in USEPA, 1988).

National Center for Food and Agricultural Policy (NCFAP). 2004. National Pesticide Use Database. Available on the Internet at: http://www.ncfap.org/database/national/default.asp. Accessed August 24, 2004.

Pavkov, K. and D. Taylor. 1988. *Rat Chronic Toxicity and Oncogenicity Study with Dyfonate. Laboratory Project ID T-11997.* (Unpublished study, as cited in USEPA, 1996).

Pena Gonzalez, P., J. Perez-Rendon Gonzalez, E. Dominguez Alberdi, M. Gomez Labat, and J. Bajo Arenas. 1996. Epidemic outbreak of acute food poisoning caused by pesticides [Article in Spanish]. *Atencion Primaria*. 17(7):467-70.

Pulsford, A. 1991. *First Amendment to a Teratology Study in CD-1 Mice with Dyfonate Technical (MRID No. 118423), Lab Project No. T-10192, T-10192C*. MRID No. 42057601. (Unpublished study as cited in USEPA, 1996).

Sauerhoff, M. 1987. *A Teratology Study in Rabbits with Dyfonate Technical*. T-l2630:Volume 1: Final Report: Laboratory Project ID: WIL 27027. (As cited in USEPA, 1996).

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

Sprague, G. and G. Zwicker. 1987. *18-Month Dietary Oncogenicity Study with Dyfonate Technical in Mice*. Final Report: T-11995. (As cited in USEPA, 1996).

Thelin, G.P. and L.P. Gianessi. 2000. *Method for Estimating Pesticide Use for County Areas of the Conterminous United States.* U.S. Geological Survey Open-File Report 00-250, 62 p. Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/ofr00250/ofr00250.pdf.

Tomlin, C.D.S. (ed). 2002. Fonofos (944-2-9). In: The e-Pesticide Manual, Version 2.2. Surrey, UK, British Crop Protection Council. (As cited in HSDB, 2004).

United States Department of Agriculture (USDA). 2003. ARS Pesticide Properties Database: Fonofos. (As cited in HSDB, 2004).

USEPA. 1988. *Fonofos Health Advisory*. EPA Report 820-K-88-003. August, 1988.

USEPA. 1991. Integrated Risk Information System (IRIS), Fonofos. March 1991. Available on the Internet at: http://www.epa.gov/iris/subst/0158.htm. Accessed February 2, 2005.

USEPA. 1992. *Pesticides in Ground Water Database: A Compilation of Monitoring Studies, 1971-1991. National Summary.* Office of Prevention, Pesticides and Toxic Substances. EPA Report 734-12-92-001. Available on the Internet at: http://nepis.epa.gov/pubtitleOPPTS.htm. [Search for document number 7341292001.]

USEPA. 1996. *Memorandum – 041701. Fonofos (Dyfonate®). Draft Toxicology Chapter for Reregistration Eligibility.* Health Effects Division. September 11, 1996.

USEPA. 1999. Reregistration Eligibility Decision (RED) Facts--O-Ethyl S-phenyl ethylphosphonodithiolate (Fonofos). EPA Report 738-F-99-019. Washington, DC: Office of Prevention, Pesticides and Toxic Substances. November 1999. Available on the Internet at: http://www.epa.gov/oppsrrd1/REDs/factsheets/0105fact.pdf.

USEPA. 2000a. *Methods for the Determination of Organic and Inorganic Compounds in Drinking Water, Volume 1*. EPA Report 815-R-00-014.

USEPA. 2000b. Method 526 - Determination of Selected Semivolatile Organic Compounds in Drinking Water by Solid Phase Extraction and Capillary Column Gas Chromatography/Mass Spectrometry (GC/MS). Revision 1.0. In: *Methods for the Determination of Organic and Inorganic Compounds in Drinking Water, Volume I*. EPA Report 815-R-00-014.

USEPA. 2000c. *Summary of Pesticide Removal/Transformation Efficiencies from Various Drinking Water Treatment Processes*. Report prepared for the Committee to Advise on Reassessment And Transition (CARAT). Available on the Internet at: http://www.epa.gov/oppfead1/carat/2000/oct/dw4.pdf.

USEPA. 2006. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

United States Geological Survey (USGS). 2004. 1997 Pesticide Use Maps. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pesticide_use_maps_1997/. Accessed August 17, 2004. Last updated August 14, 2003.

Wauchope, R.D., T.M. Buttler, A.G. Hornsby, P.W.M. Augustijn-Beckers, and J.P. Burt. 1992. The SCS/ARS/CES Pesticide Properties Database for Environmental Decision-Making. *Reviews of Environment Contamination and Toxicology.* 123:1-157. (As cited in Extoxnet, 1993).

Windholz, M., S. Budavari, R.F. Blumetti, and E.S. Otterbein (eds.) 1983. *The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals.* 10th ed. Rahway, NJ: Merck and Co., Inc.

Woodard, M.W., C.L. Leigh, and G. Woodard. 1968. *Dyfonate (N-2790) Three-Generation Reproduction Study in Rats*. (Unpublished study, as cited in USEPA, 1996).

Woodard, M.W., C.I. Donoso, and J.P. Gray, et al. 1969. *Dyfonate (N-2790) Safety Evaluation by Dietary Administration to Dogs for 106 Weeks*. (Unpublished study, as cited in USEPA, 1988 and USEPA, 1996).

Yalkowsky, S.H. and Y. He. (eds.) 2003. *Handbook of aqueous solubility data*. Boca Raton, FL: CRC Press. (As cited in HSDB, 2004).

Chapter 10: Terbacil

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

Terbacil, a synthetic organic compound (SOC), is a selective herbicide used to control broadleaf weeds and grasses on terrestrial food/feed crops (e.g., apples, mint, peppermint, spearmint, and sugarcane), terrestrial food (e.g., asparagus, blackberry, boysenberry, dewberry, loganberry, peach, raspberry, youngberry, and strawberry), terrestrial feed (e.g., alfalfa, forage, and hay) and forest trees (e.g., cottonwood).

Terbacil is a persistent and potentially mobile herbicide in terrestrial environments. Because of its low affinity to soils, it can potentially leach into ground and/or surface waters.

In acute and subchronic toxicity studies, terbacil is practically non-toxic. Terbacil is not considered to be a developmental or reproductive toxicant. Terbacil shows no evidence of carcinogenicity and is considered unlikely to be carcinogenic to humans (Group E). Terbacil is not mutagenic.

In chronic dietary exposure studies in animals, the liver is a primary target organ. The reference dose (RfD) of 0.013 mg/kg/day for terbacil is calculated from a two-year chronic study in beagle dogs. The lowest-observed-adverse-effect level (LOAEL) of 6.25 mg/kg/day was based on increased thyroid-to-body weight ratios, slight increases in liver weights, and elevated alkaline phosphatase levels with a no-observed-adverse-effect level (NOAEL) of 1.25 mg/kg/day. In deriving the RfD, the Agency applied an uncertainty factor of 100 to account for interspecies and intraspecies differences. Using the RfD of 0.013 mg/kg/day and applying a 20 percent screening relative source contribution, the Agency derived a health reference level (HRL) of 0.090 mg/L (or 90 µg/L) for terbacil.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. In the case of terbacil, the Agency determined that the RfD is adequately protective of children. No other potentially sensitive subpopulation has been identified.

In 1998, EPA estimated that agricultural usage consumed approximately 221,000 to 447,000 pounds of terbacil annually and non-agricultural usage consumed approximately 9,000 to 14,000 pounds. These estimates are based on data collected mostly between 1990 and 1995, and in some cases as early as 1987. According to the National Center for Food and Agricultural Policy (NCFAP), approximately 298,000 pounds of terbacil were applied annually in agriculture around 1992 and approximately 342,000 pounds were applied around 1997.

The Toxic Release Inventory (TRI) provides information on industrial releases of terbacil. Data are reported from a single State, Texas, for the time period covering 1995 to 1997. During this three-year period, all reported releases were on-site releases to surface water; these releases varied between 3,000 to 10,000 pounds annually.

Data on the ambient occurrence of terbacil are available from the first monitoring cycle (1992-2001) of the United States Geological Survey's (USGS's) National Ambient Water Quality Assessment (NAWQA) program. While the USGS detected terbacil in both surface and

ground waters, in all land use settings the 95th-percentile concentration was less than 0.034 µg/L (the USGS reporting limit). The maximum surface water concentration, 0.54 µg/L (found in an agricultural setting), and the maximum ground water concentration, 0.891 µg/L (found in a mixed land use setting), are both less than the HRL and ½ the HRL.

In order to determine the extent of terbacil contamination of drinking water, EPA included terbacil as an analyte in the first Unregulated Contaminant Monitoring Regulation (UCMR 1). None of the 3,866 public water systems (PWSs) sampled (serving a total population of 226 million) had detections of terbacil at or above the minimum reporting level (MRL) of 2 µg/L. These data indicate that no occurrence and exposure is expected at levels greater than the HRL (90 µg/L) or even ½ the HRL (45 µg/L).

EPA also evaluated several sources of supplemental information on terbacil occurrence in drinking water. In the National Pesticide Survey, which collected samples from approximately 1,300 community water systems and rural drinking water wells between 1988 and 1990, terbacil was not detected (using a minimum reporting limit of 1.7 µg/L). The Pesticides in Ground Water Database indicates that terbacil was found in 6 of 288 ground water wells in 6 States. Terbacil was found in 1 ground water well in Oregon (at a concentration of 8.9 µg/L) and 5 ground water wells in West Virginia (with concentrations ranging from 0.3 to1.2 µg/L). None of the detections exceeded the HRL or ½ the HRL.

The Agency has made a preliminary determination not to regulate terbacil with a national primary drinking water regulation (NPDWR). Because terbacil does not appear to occur at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

a.i.	Active Ingredient
AOAC	Association of Official Analytical Chemists
APHA	American Public Health Association
ASTM	American Society for Testing and Materials
CAS	Chemical Abstracts Service
CCL 2	Second Contaminant Candidate List
CWS	Community Water System
DNA	Deoxyribonucleic Acid
FQPA	Food Quality Protection Act
GAC	Granular Activated Carbon
GC	Gas Chromatography
GC/MS	Gas Chromatography with Mass Spectrometry
GC/NPD	Gas Chromatography with a Nitrogen-Phosphorus Detector
HRL	Health Reference Level
LLE	Liquid-Liquid Extraction
LOAEL	Lowest Observed Adverse Effect Level
LSE	Liquid-Solid Extraction
MDL	Method Detection Limit
MRL	Minimum Reporting Level
MTBE	Methyl tertiary-butyl ether
NAWQA	National Water Quality Assessment
NCFAP	National Center for Food and Agricultural Policy
NOAEL	No Observed Adverse Effect Level
NPD	Nitrogen-Phosphorus Detector
NPDWR	National Primary Drinking Water Regulation
NPS	National Pesticide Survey
NTNCWS	Non-Transient Non-Community Water System
PGWDB	Pesticides in Ground Water Database
PWS	Public Water System
RfD	Reference Dose
RL	Reporting Limit
TRI	Toxics Release Inventory
UCMR 1	First Unregulated Contaminant Monitoring Regulation
USGS	United States Geological Survey

10 Terbacil

10.1 Definition

Terbacil is a synthetic organic compound, specifically a substituted uracil. The Chemical Abstracts Service (CAS) name for terbacil is 3-*tert*-butyl-5-chloro-6-methyluracil, and its registry number is 5902-51-2. Terbacil's trade names include Sinbar (most common), DuPont 732, and Geonter. In the United States, it is manufactured in Delaware by DuPont Agricultural Products.

10.1.1 Properties and Sources

Terbacil is an odorless, white crystalline solid, most often available as a wettable powder (Extoxnet, 1994). As a selective herbicide, it acts by inhibiting photosynthesis. Terbacil is used to control annual and perennial grasses and broad-leaf weeds in agricultural fields and fruit and nut orchards. Occasionally, terbacil will be found in mixed formulations with other herbicides. As a synthetic compound, it does not occur naturally. Some physical and chemical properties of terbacil are listed in Exhibit 10-1.

Exhibit 10-1: Physical and Chemical Properties of Terbacil

Identification	
CAS number	5902-51-2
Molecular Formula	$C_9H_{13}ClN_2O_2$
Physical and Chemical Properties	
Boiling Point	-----
Melting Point	175 - 177 °C [1]
Molecular Weight	216.67 g/mol [1]
Log K_{oc}	1.64 - 1.93 [2]
Log K_{ow}	1.89 [3]
Water Solubility	710 mg/L at 25 °C [4]
Vapor Pressure	4.7 x 10^{-7} mm Hg at 29.5 ° C [5]
Henry's Law Constant	2.7 x 10^{-10} atm-m^3/mole [2] 7.8 x 10^{-9} (dimensionless), predicted [6] 4.8 x 10^{-9} (dimensionless), from literature [6]
Freundlich Isotherm Constant (K)	69,300 (µg/g)(L/µg)$^{1/n}$ [6]

[1] *Budavari, 1996 (as cited in HSDB, 2004)*

[2] *HSDB, 2004*

[3] *Hansch et al., 1995 (as cited in HSDB, 2004)*

[4] *Tomlin, 1997 (as cited in HSDB, 2004)*

[5] *Ahrens, 1994 (as cited in HSDB, 2004)*

[6] *Speth et al., 2001*

10.1.2 Environmental Fate and Behavior

Because terbacil is applied to fields and orchards by ground or aerial spraying, it can contaminate the air, soil, and water. Terbacil is persistent, slow to degrade, and potentially very mobile in the environment. It can undergo photodegradation in surface soil, but this process is slow, with a calculated half-life of 122 days (Barefoot, 1986 as cited in USEPA, 1998). Terbacil's aerobic and anaerobic half-lives in soil have been measured at 653 days and 235 days, respectively (Atkins *et al.*, 1992a, 1992b both as cited in USEPA, 1998). Thus, in some cases, terbacil can remain in the soil for more than one growing season. The persistence of terbacil varies depending on the application rate, soil type, availability of oxygen, and rainfall (Extoxnet, 1994). Field dissipation studies found terbacil half-lives of 204 days on silty clay soil, 212 days on silt loam soil, and 252 days on sandy loam soil (Dupont, 1995 as cited in USEPA, 1998). Where soil moisture is adequate, terbacil is subject to microbial degradation, although few data are available on the degradation rates and products.

Terbacil exhibits low affinity for adsorption to soil (see Exhibit 10-1). This characteristic, combined with moderate water solubility, explains its mobility in the environment. Terbacil is capable of leaching through the soil column and contaminating ground water. Leaching appears to occur more readily in sandy or coarse soils than in organic or fine-textured soils (Extoxnet, 1994). In the field dissipation studies mentioned above (DuPont, 1995 as cited in USEPA, 1998), aerially applied terbacil was later detected in the soil at depths of up to 45 to 50 cm (USEPA, 1998). In laboratory tests, terbacil leached through several different 30 cm soil columns, and was detected primarily in the leachate (Atkins, 1992c as cited in USEPA, 1998). Also observed in the leachate were several terbacil degradates, including t-butylurea, 3-t-butyl-6-methyluracil, and 6-chloro-2,3-dihydro-7-(hydroxymethyl)-3,3-dimethyl-5H-oxazolo(3,2-a)pyrimidin-5-one.

Terbacil can enter surface waters either directly through deposition of spray drift from application or indirectly in runoff from treated soil. It appears to be stable to abiotic hydrolysis at a wide range of pH values, but is susceptible to slow photodegradation, with a half-life of 29 days in standard reference water under natural sunlight (Rhodes, 1975 as cited in USEPA, 1998). If the water contains suspended sediments or particulate material, the rate of photolysis is even slower. Certain compounds, such as methylene blue or riboflavins, act as photosensitizers and can accelerate the light-mediated decomposition process. Modeling studies conducted by EPA suggest that terbacil may accumulate in the range of 28 to 1,470 µg/L in surface water and up to 125 µg/L in ground water (USEPA, 1998). There are currently no data on aerobic metabolism of terbacil in water.

Terbacil has a low vapor pressure and a low Henry's constant, and thus is unlikely to be found in significant concentrations in the atmosphere (USEPA, 1998).

10.2 Health Effects

In acute and subchronic toxicity studies, terbacil is practically non-toxic (Haskell Laboratories, 1965a, 1965b both as cited in USEPA, 1998). Terbacil does not cause dermal sensitivity in rabbits or guinea pigs and causes mild conjunctival eye irritation in rabbits (Henry, 1986; Hood, 1966 both as cited in USEPA 1998). In rats exposed subchronically to dietary terbacil, effects were seen at a "lowest-observed-adverse-effect level" (LOAEL) of 25 mg/kg/day and included increased absolute and relative liver weights, vacuolization, and enlargement of liver cells (Wazeter *et al.*,1964; Haskell Laboratories, 1965c both as cited in USEPA, 1998).

A primary target organ in rats following exposure to terbacil is the liver. Chronic effects of dietary terbacil exposure in two-year studies included increases in thyroid-to-body weight ratios, slight increases in liver weights and elevated alkaline phosphatase levels in beagle dogs, significant decreases in body weight in rats, increases in serum cholesterol levels and increases in liver to body weight ratios in rats (Wazeter *et al.*,1967a; Malek, 1993 both as cited in USEPA, 1998). In beagle dogs, effects were seen at or above 6.25 mg/kg/day ("no-observed-adverse-effect level" [NOAEL] = 1.25 mg/kg/day). In rats, effects (i.e., decreases in body weight, increases in liver weights and cholesterol levels) were seen at higher levels (LOAELs = 56 mg/kg/day for males and 83 mg/kg/day for females).

Terbacil is not considered to be a developmental or reproductive toxicant. In developmental studies, maternal effects were generally seen prior to or at the same levels as developmental effects. Haskell Laboratories (1980 as cited in USEPA, 1998) reported maternal effects (i.e., decreased body weight) and significant decreases in the number of live fetuses per litter due to early fetal resorption at a LOAEL of 62.5 mg/kg/day in rats. In rabbits administered terbacil via gavage, the maternal and developmental LOAELs were equal (600 mg/kg/day). Maternal toxicity was based on the death of the dams and developmental toxicity was based on a decrease in live fetal weights (Solomon, 1984 as cited in USEPA, 1998). No reproductive effects were seen in a three-generation study where terbacil was administered to male and female rats at dose levels of 2.5 and 12.5 mg/kg/day (Wazeter *et al.*, 1967b as cited in USEPA, 1998).

Terbacil is not mutagenic. Terbacil was tested and found negative in a chromosomal aberration study in rat bone marrow cells, found negative in a gene mutation assay (with and without S9 activation), and found negative for deoxyribonucleic acid (DNA) synthesis when tested up to cytotoxic levels in rats (Cortina, 1984; Haskell Laboratories, 1984 as cited in USEPA, 1998). Terbacil shows no evidence of carcinogenicity and is unlikely to be carcinogenic to humans (Group E) (USEPA, 1998).

The reference dose (RfD) of 0.013 mg/kg/day for terbacil (USEPA, 1998) is calculated from a two-year chronic study in beagle dogs. The LOAEL of 6.25 mg/kg/day was based on increased thyroid-to-body weight ratios, slight increases in liver weights, and elevated alkaline phosphatase levels with a NOAEL of 1.25 mg/kg/day. In deriving the RfD, the Agency applied an uncertainty factor of 100 to account for interspecies and intraspecies differences. Using the RfD of 0.013 mg/kg/day and applying a 20 percent screening relative source contribution, the Agency derived a health reference level (HRL) of 0.090 mg/L (or 90 µg/L) for terbacil.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. In the case of terbacil, the Agency determined that there was no need to apply a Food Quality Protection Act (FQPA) factor to the RfD in order to protect children (USEPA, 1998). Other potentially sensitive subpopulations have not been identified.

10.3 Occurrence and Exposure

10.3.1 Use and Environmental Release

Terbacil (3-tert-butyl-5-chloro-6-methyl uracil) is manufactured by E.I. DuPont de Nemours and Company, Inc. It is marketed under several trade names, including Sinbar, DuPont Herbicide 732, and Geonter. Terbacil is currently registered for use as an herbicide on terrestrial food and feed crops, including apple, mint, sugarcane, asparagus, blackberry, boysenberry, dewberry, loganberry, peach, raspberry, youngberry, and strawberry, as well as for applications on ornamentals and in forestry, particularly for cottonwoods. Terbacil is not currently registered for residential use. Terbacil is generally applied by spraying, either from tractor-mounted booms or from aircraft (USEPA, 1998).

EPA has estimated that agricultural usage of terbacil consumes approximately 221,000 to 447,000 pounds of active ingredient (a.i.) annually, and non-agricultural usage consumes

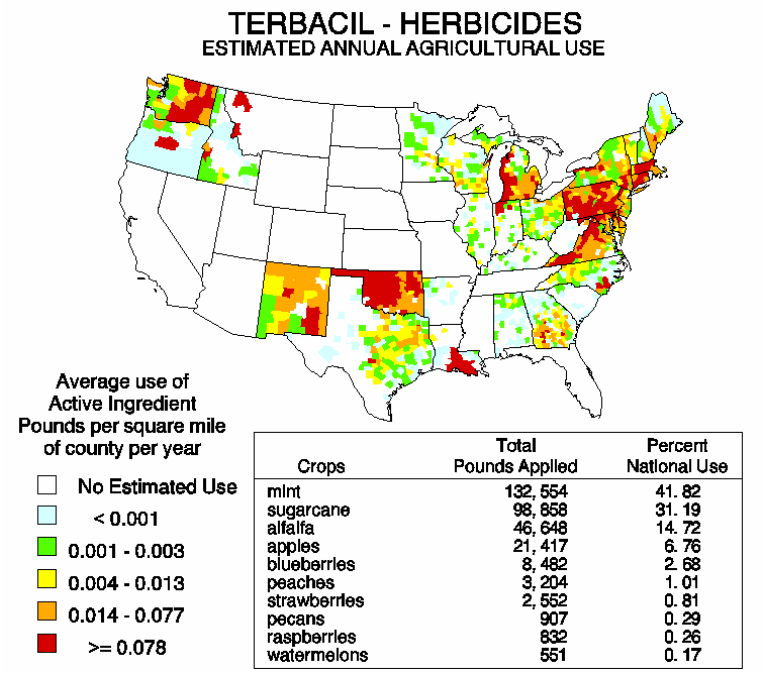
approximately 9,000 to 14,000 pounds. These estimates are based on data collected mostly between 1990 and 1995, and in some cases as early as 1987 (USEPA, 1998).

The National Center for Food and Agricultural Policy (NCFAP) estimates of national agricultural terbacil use indicate an increase during the 1990s. According to NCFAP, around 1992 approximately 298,000 pounds of terbacil a.i. were applied annually to 12 types of crops on 353,000 acres, and in 1997 approximately 342,000 pounds a.i. were applied annually to 13 types of crops on 357,000 acres. NCFAP estimates are based on State-level commercial agriculture usage patterns for the periods 1990-1993 and 1995-1998, and State-level crop acreage for 1992 and 1997 (NCFAP, 2004). For more information on NCFAP pesticide use estimates, see Chapter 2.

The United States Geological Survey (USGS) combined data collected by NCFAP with data from the Census of Agriculture to estimate that 285,000 pounds of terbacil a.i. per year were used on approximately 4.0 million agricultural acres in the early 1990s (Thelin and Gianessi, 2000). While USGS has not published national estimates for 1997, an estimate of approximately 317,000 pounds a.i. can be inferred from the "total pounds applied" and "percent national use" data in the 1997 geographical distribution map (Exhibit 10-2).

Exhibit 10-2 shows the estimated geographic distribution and intensity of typical annual terbacil use in the United States in the late 1990s. A breakdown of use by crop is also included. The map was created by USGS using State-level data sets on pesticide use rates from 1995-1998 compiled by NCFAP, combined with county-level data on harvested crop acreage obtained from the 1997 Census of Agriculture (USGS, 2004). Due to the nature of the data sources, non-agricultural uses are not reflected on the map and variations in use at the county-level are also not well represented (Thelin and Gianessi, 2000). For background on the USGS pesticide use maps, see Chapter 2. The map indicates that terbacil is commonly used in the Pacific Northwest, the Northeast, the Great Lakes Region, and parts of the South and Southwest.

Exhibit 10-2: Estimated Annual Agricultural Use of Terbacil, c. 1997



Crops	Total Pounds Applied	Percent National Use
mint	132,554	41.82
sugarcane	98,858	31.19
alfalfa	46,648	14.72
apples	21,417	6.76
blueberries	8,482	2.68
peaches	3,204	1.01
strawberries	2,552	0.81
pecans	907	0.29
raspberries	832	0.26
watermelons	551	0.17

Source: USGS, 2004

Terbacil is listed as a Toxics Release Inventory (TRI) chemical. TRI data for terbacil (see Exhibit 10-3) are reported for the years 1995 to 1997. During that three-year period, all reported releases were on-site releases to surface water. These releases were all in Texas (USEPA, 2006a). For a discussion of the limitations of TRI data, see Chapter 2.

Exhibit 10-3: Environmental Releases (in pounds) of Terbacil in the United States, 1995-1997

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1995	0	4,608	0	0	0	4,608
1996	0	3,835	0	0	0	3,835
1997	0	10,318	0	0	0	10,318

Source: USEPA, 2006a

10.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are the source of most drinking water. Data on the occurrence of terbacil in ambient surface and ground water are available from the National Water Quality Assessment (NAWQA) program of the USGS. For details on this program, see the discussion in Chapter 2. NAWQA data have been analyzed independently by USGS and EPA.

NAWQA National Pesticide Synthesis

Under the NAWQA program, USGS monitored terbacil between 1992 and 2001 in representative watersheds and aquifers across the country. Reporting limits varied but did not exceed 0.034 µg/L. All concentrations determined for terbacil are estimated concentrations.

In surface water (Exhibit 10-4), terbacil was detected at frequencies ranging from 1.40% of samples in undeveloped settings to 1.82% in mixed land use settings, 1.98% in urban settings, and 4.52% in agricultural settings. The 95th percentile concentrations were less than the reporting limit in all settings. The highest concentration, 0.540 µg/L, was found in an agricultural setting (Martin *et al.*, 2003).

Exhibit 10-4: USGS National Synthesis Summary of NAWQA Monitoring of Terbacil in Ambient Surface Water, 1992-2001

Land Use Type	No. of Samples (and No. of Sites)	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,858 (77)	4.52%	<RL	<RL	0.540 µg/L
Mixed	996 (46)	1.82%	<RL	<RL	0.341 µg/L
Undeveloped	60 (4)	1.40%	<RL	<RL	0.092 µg/L
Urban	896 (33)	1.98%	<RL	<RL	0.035 µg/L

Notes:
RL = Reporting limit. Reporting limits for terbacil varied, but did not exceed 0.034 µg/L.

All terbacil concentrations are estimated concentrations.

The USGS Pesticide National Synthesis used one year of data, generally the year with the most sampling results, to represent each site in this analysis. The sampling results were time-weighted, to eliminate bias from more frequent sampling at certain times of year. Detection Frequencies and Percentile Concentrations can be interpreted as representing annual occurrence. For instance, the detection frequency can be thought of as the percent of the year in which detections are found at a typical site in this land use category, and the 95th percentile concentration can be thought of as a concentration that is not exceeded for 95% of the year at a typical site in this land use category.

Source: Martin et al., 2003

In ground water (Exhibit 10-5), terbacil detection frequencies ranged from 0.0% in undeveloped settings to 0.26% in mixed land use (major aquifer) settings, 0.76% in agricultural settings, and 1.20% in urban land use settings. The 95th percentile concentrations were less than the reporting limit in all settings. The highest concentration, 0.891 µg/L, was in a mixed land use (major aquifer) setting (Kolpin and Martin, 2003).

Exhibit 10-5: USGS National Synthesis Summary of NAWQA Monitoring of Terbacil in Ambient Ground Water, 1992-2001

Land Use Type	No. of Wells	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,438	0.76%	<RL	<RL	0.495 µg/L
Mixed (Major Aquifer)	2,708	0.26%	<RL	<RL	0.891 µg/L
Undeveloped	67	0.0%	<RL	<RL	<RL
Urban	830	1.20%	<RL	<RL	0.093 µg/L

Notes:
RL = Reporting limit. Reporting limits for terbacil varied, but did not exceed 0.034 µg/L.

All terbacil concentrations are estimated concentrations.

The USGS Pesticide National Synthesis considered each well a distinct site in this analysis. Each well was represented by one sample: normally the first one taken, but possibly a later sample if the first sample was not analyzed for the full range of analytes.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Kolpin and Martin, 2003

EPA Summary Analysis of NAWQA Data

Whereas the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination, including terbacil. Detection frequencies were simply computed as the percentage of samples and sites with detections (i.e., with at least one result equal to or greater than the reporting limit). Note that reporting limits were not uniform. Sample detections can be biased by frequent sampling in areas with high (or low) occurrence. Calculating the percentage of sites with detections can reduce this bias. For more details on the data set and the EPA analysis, see Chapter 2.

The results of the EPA analysis are presented in Exhibit 10-6. Overall, terbacil was detected in 2.7% of samples and at 2.8% of sites. Terbacil was detected more frequently in surface water than in ground water. Although the highest concentration (1.52 µg/L) was found in surface water, in general ground water concentrations tended to be higher than surface water concentrations.

Exhibit 10-6: EPA Summary Analysis of Terbacil Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	14,885	3.6%	1,900	8.5%	0.0021	0.0215	0.208	0.72	1.52
ground water	6,355	0.7%	5,200	0.7%	0.003	0.0273	0.891	1.05	1.05
all sites	21,240	2.7%	7,100	2.8%	0.0021	0.0219	0.260	0.921	1.52

[1] RLs (Reporting Limits) for terbacil varied but did not exceed 0.034 µg/L. For more information, see Chapter 2. Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible.

10.3.3 Drinking Water Occurrence

Nationally representative data on terbacil occurrence in drinking water have been collected by large and small public water systems in accordance with EPA's first Unregulated Contaminant Monitoring Regulation (UCMR 1). For a complete description of the UCMR, see Chapter 2 and USEPA (2006b). In addition, historical data are available from the Pesticides in Ground Water Database.

UCMR 1

UCMR 1 monitoring was conducted primarily between 2001 and 2003, though some results were not collected and reported until as late as 2005. As a List 1 contaminant, terbacil was scheduled to be monitored by all large community water systems (CWSs) and non-transient non-community water systems (NTNCWSs) and a statistically representative sample of small CWSs and NTNCWSs. The data presented in this report reflect UCMR 1 analytical samples submitted and quality-checked under the regulation as of July 2005. Terbacil data were collected and submitted by 797 (99.6 percent) of the 800 small systems selected for the small system sample and 3,069 (99.0 percent) of the 3,100 large systems defined as eligible for the UCMR 1 large system census. Terbacil data have been analyzed at the level of simple detections (at or above the minimum reporting level, ≥ MRL, or ≥ 2 µg/L), exceedances of the health reference level (> HRL, or > 90 µg/L), and exceedances of one-half the value of the HRL (> ½ HRL, or > 45 µg/L).

Results of the analysis are presented in Exhibits 10-7 and 10-8. No detections of terbacil were found in any samples, and thus there were also no exceedances of the HRL or one-half the HRL.

Exhibit 10-7: Summary UCMR 1 Occurrence Statistics for Terbacil in Small Systems (Based on Statistically Representative National Sample of Small Systems)

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,251		--
Percent of Samples with Detections	0.00%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	90 µg/L		--
Minimum Reporting Level (MRL)	2 µg/L		--
Maximum Concentration of Detections	< MRL		--
99th Percentile Concentration of Detections	< MRL		--
Median Concentration of Detections	< MRL		--
Total Number of PWSs	797		60,414
Number of GW PWSs	590		56,072
Number of SW PWSs	207		4,342
Total Population	2,760,570		45,414,590
Population of GW PWSs	1,939,815		36,224,336
Population of SW PWSs	820,755		9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%	0
PWSs (GW & SW) > 1/2 HRL	0	0.00%	0
PWSs (GW & SW) > HRL	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	0	0.00%	0
Population Served by PWSs > 1/2 HRL	0	0.00%	0
Population Served by PWSs > HRL	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 10-8: Summary UCMR 1 Occurrence Statistics for Terbacil in Large Systems (Based on the Census of Large Systems)

Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	30,386	
Percent of Samples with Detections	0.00%	
99th Percentile Concentration (all samples)	< MRL	
Health Reference Level (HRL)	90 µg/L	
Minimum Reporting Level (MRL)	2 µg/L	
Maximum Concentration of Detections	< MRL	
99th Percentile Concentration of Detections	< MRL	
Median Concentration of Detections	< MRL	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	3,069 1,375 1,694	
Total Population Population of GW PWSs Population of SW PWSs	223,361,341 53,303,000 170,058,341	
Occurrence by System	**Number**	**Percentage**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%
PWSs (GW & SW) > 1/2 HRL	0	0.00%
PWSs (GW & SW) > HRL	0	0.00%
Occurrence by Population Served		
Population Served by PWSs with Detections	0	0.00%
Population Served by PWSs > 1/2 HRL	0	0.00%
Population Served by PWSs > HRL	0	0.00%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, and PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark; Population Served by PWSs with detections, by PWSs > ½ HRL, and by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-The HRL used in this analysis is a draft value for working review only.

Summary Analysis of Combined Large and Small System UCMR Data

None of the 3,866 public water systems (PWSs) sampled (serving a population of 226 million) had detects for terbacil at the MRL of 2 µg/L. Hence, these data indicate that no occurrence and exposure is expected at levels greater than 45 µg/L (½ the HRL) and greater than 90 µg/L (the terbacil HRL).

Pesticides in Ground Water Database (PGWDB)

The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992). Most of the data are from drinking water wells. Since PGWDB data come from multiple sources, they should be interpreted with caution. Results might be biased high, because areas with suspected contamination are likely to have been sampled more frequently than pristine areas. For more information on PGWDB, see Chapter 2.

According to the data compiled in the PGWDB, terbacil was detected in 6 (2.08 percent) of 288 wells sampled. The detections were found in 2 out of 6 States where terbacil was investigated. Terbacil was found in one ground water well in Oregon (at a concentration of 8.9 µg/L) and five ground water wells in West Virginia (with concentrations ranging from 0.3 to1.2 µg/L). All detections were well below the HRL of 90 µg/L (USEPA, 1992).

National Pesticide Survey (NPS)

EPA collected samples from approximately 1,300 CWS wells and rural drinking water wells between 1988 and 1990 for the National Pesticide Survey (NPS). The survey was designed to provide a statistically reliable estimate of pesticide occurrence in the nation's drinking water wells. For details about NPS, see Chapter 2.

With a minimum reporting limit of 1.7 µg/L, terbacil was not detected in the survey (USEPA, 1990).

10.4 Technology Assessment

10.4.1 Analytical Methods

EPA evaluated the availability of analytical methods for all of the unregulated contaminants considered for UCMR 1 in 1999 (64 FR 50556). Sources for these methods included publications by EPA and by voluntary consensus standard organizations such as the American Society for Testing and Materials (ASTM), the Association of Official Analytical Chemists (AOAC), and the American Public Health Association (APHA).

Terbacil is a UCMR List 1 contaminant that can be detected in drinking water using EPA Methods 507 and 525.2. These methods were approved for the monitoring of terbacil in 1999 (64 FR 50556). EPA Method 507 relies on liquid-liquid extraction (LLE) of the method analytes, followed by gas chromatography with a nitrogen-phosphorus detector (GC/NPD), while EPA Method 525.2 relies on liquid-solid extraction (LSE) and capillary column gas chromatography with mass spectrometry (GC/MS). A full description of both EPA Methods can be found in EPA's *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3* (USEPA, 1995a). Additional methods approved for terbacil include ASTM Method D5475-93 (ASTM, 1996; 1998) and AOAC International 991.07 (AOAC, 1998).

The method detection limit (MDL) and the average recovery for each analytical method that can be used for the analysis of terbacil in water are included in the method descriptions below.[1]

EPA Method 507

In EPA Method 507 (Revision 2.1), *"Determination of Nitrogen and Phosphorus-Containing Pesticides in Water by Gas Chromatography with a Nitrogen-Phosphorus Detector,"* approximately 1 liter of sample is extracted with methylene chloride by shaking in a separatory funnel. The methylene chloride extract is separated, dried, and concentrated during a solvent exchange to methyl *tert*-butyl ether (MTBE). Chromatographic conditions are set to allow for separation and measurement of the analytes in the extract by capillary column gas chromatography (GC) with a nitrogen-phosphorus detector (NPD) (USEPA, 1995b).

The MDL for terbacil is reported as 0.56 µg/L, and the average recovery is reported to range from 86 to 102 percent depending on the method option used (USEPA, 1995b).

EPA Method 525.2

In EPA Method 525.2 (Revision 2.0), *"Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry*," analytes are extracted by LSE: a water sample is passed through a disk or cartridge containing a solid matrix with a chemically bonded C_{18} organic phase. The organic compounds are eluted from the LSE disk or cartridge with small amounts of ethyl acetate and methylene chloride. The analytes are then concentrated by evaporation of some of the solvent. The concentrated sample extract is analyzed by injecting an aliquot onto a capillary GC column. Compounds eluting from the GC column are characterized by comparing their measured mass spectra and retention times to reference mass spectra and retention times (USEPA, 1995c).

The MDL for terbacil is reported to range from 0.22 to 2.1 µg/L, and the average recovery is reported to range from 97 to 129 percent, depending on the method option used (USEPA, 1995c).

[1] The Method Detection Limit (MDL) is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon the precision of a series of replicate measurements of the analyte at low concentrations. The MDL incorporates estimates of the accuracy of the determination. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

The average recovery is the fraction or percent concentration of a target analyte determined relative to the true or expected concentration from a sample containing a known amount of the target analyte. (This can result in apparent recovery values greater than 100 percent.)

10.4.2 Treatment Technologies

Treatment technology status does not influence the determination of whether or not a contaminant should be regulated. However, treatment technologies must be readily available before a contaminant can be regulated with a national primary drinking water regulation (NPDWR). There is no evidence that terbacil is substantially removed by conventional treatments, such as coagulation/flocculation, sedimentation, and inert media filtration. Reverse osmosis is effective in removing many synthetic organic chemicals, but no specific data are available for terbacil removal. Currently, the most viable known treatment technology is activated carbon.

Granular activated carbon (GAC) treatment removes contaminants via the physical and chemical process of sorption: the contaminants attach to the carbon surface as water passes through the carbon bed. Activated carbon has a large sorption capacity for many water impurities, including synthetic organic chemicals, taste- and odor-causing compounds, and some species of mercury.

Adsorption capacity is typically represented by the Freundlich isotherm constant, with higher Freundlich (K) values indicating greater sorption potential. Activated carbon is considered to be cost-effective for removing a particular contaminant if the Freundlich (K) value of the contaminant is above 200 $\mu g/g\ (L/\mu g)^{1/n}$ (Speth *et al.*, 2001). Speth *et al.* (2001) report that the Freundlich (K) value for terbacil is 69,300 $\mu g/g\ (L/\mu g)^{1/n}$, which indicates that GAC is a promising treatment option.

10.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate terbacil with an NPDWR. Because terbacil does not appear to occur at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. Terbacil has been found in ambient waters but the levels were less than the HRL (as well as ½ the HRL). It was not found in the UCMR 1 survey of public water supplies.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

10.6 References

Ahrens, W. 1994. *Herbicide Handbook of the Weed Science Society of America*. 7th Edition, Weed Science Society of America, Champaign, IL. (As cited in HSDB, 2004).

American Society for Testing and Materials (ASTM). 1996. *Annual Book of ASTM Standards.* Volume 11.02. West Conshohocken, PA: American Society for Testing and Materials.

ASTM. 1998. *Annual Book of ASTM Standards.* Volume 11.02. West Conshohocken, PA: American Society for Testing and Materials.

Association of Official Analytical Chemist (AOAC) International. 1998. *Official Methods of Analysis of AOAC International.* 16th ed. 4th Revision. Volume I. Baltimore, MD: AOAC International.

Atkins, R., D. Perdue, and A. Kesterson. 1992a. *Aerobic Soil Metabolism of [carbon 14]-Terbacil*. Lab Project Number: AMR 1689-90: 1403: 449. (Unpublished study, as cited in USEPA, 1998).

Atkins, R., D. Perdue, and A. Kesterson. 1992b. *Anaerobic Soil Metabolism of [carbon 14] Terbacil*. Lab Project Number: AMR1688-90: 1392: 450. (Unpublished study, as cited in USEPA, 1998).

Atkins, R., D. Perdue, and A. Kesterson. 1992c. *Aged Soil Column Leaching of [carbon 14] Terbacil.* Lab Project Number: AMR 2154-91: 1396: 597 (Unpublished study, as cited in USEPA, 1998).

Barefoot, A. 1986. *Photolysis of Terbacil on Soil*. Document No. AMR-521-86. (Unpublished study, as cited in USEPA, 1998).

Budavari, S. (ed.). 1996. *The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals*. Whitehouse Station, NJ: Merck & Co., Inc. (As cited in HSDB, 2004).

Cortina, T. 1984. *In vivo Bone Marrow Chromosome Study in Rats: H#14,673.* Final Report: Project No. 201-695. (Unpublished study, as cited in USEPA, 1998)

DuPont Agricultural Products (DuPont). 1995. Submission of Environmental Data in Support of the Terbacil Registration Standard. Transmittal of 1 Study. MRID 43585500. (As cited in USEPA, 1998).

Extoxnet. 1994. "Extension Toxicology Network Pesticide Information Profiles–Terbacil." Available on the Internet at: http://pmep.cce.cornell.edu/profiles/Extoxnet/pyrethrins-ziram/terbacil-ext.html. Accessed March 10, 2006.

Hansch, C., A.J. Leo, and D. Hoekman. 1995. *Exploring QSAR – Hydrophobic, Electronic, and Steric Constants*. Washington, DC: American Chemical Society. (As cited in HSDB, 2004).

Haskell Laboratories. 1965a. *14-day Rat Feeding Study*. (Unpublished study, as cited in USEPA, 1998).

Haskell Laboratories. 1965b. *3-week Rabbit Dermal Toxicity Study*. (Unpublished study, as cited in USEPA, 1998).

Haskell Laboratories. 1965c. *Rat 90-day feeding study in Rats*. (Unpublished study, as cited in USEPA, 1998).

Haskell Laboratories. 1980. *Rat Oral Teratology with Terbacil*. (Unpublished study, as cited in USEPA, 1998).

Haskell Laboratories. 1984. *CHO (HGPRT) Gene Mutation Assay with Terbacil*. (Unpublished study, as cited in USEPA, 1998).

Hazardous Substances Data Bank (HSDB). 2004. Search for Terbacil. Available on the internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed November 1, 2004. Last updated May 4, 2001.

Henry, J. 1986. *Skin Sensitization Test of Terbacil (IND-732-53) in Guinea Pigs for EPA Pesticide Registration.* Revised. Report No. 600-85: MR No. 4581-277. (As cited in USEPA, 1998).

Hood, D. 1966. *15-exposure Skin Absorption Studies with 3-tert-Butyl-5-chloro-6-methyluracil*. Report No. 33-66. (As cited in USEPA, 1998).

Kolpin, D.W. and J.D. Martin. 2003. "Pesticides in Ground Water: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestgw/Pest-GW_2001_Text.html. Accessed August 24, 2004.

Malek, D. 1993. *Combined Chronic Toxicity/Oncogenicity Study with DPX-D732-66 (Terbacil) Two Year Feeding Study in Rats*. Lab Project Number: 9027-001: 453-93: 18260. (Unpublished study, as cited in USEPA, 1998).

Martin, J.D., C.G. Crawford, and S.J. Larson. 2003. "Pesticides in Streams: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestsw/Pest-SW_2001_Text.html. Accessed August 24, 2004.

National Center for Food and Agricultural Policy (NCFAP). 2004. National Pesticide Use Database. Available on the Internet at: http://www.ncfap.org/database/default.htm.

Rhodes, R.C. 1975. Biodegradation *Studies with 2-14C-Terbacil in Water and Soil*. (Unpublished study, as cited in USEPA, 1998).

Solomon, H. 1984. *Embryo-Fetal Toxicity and Teratogenicity Study of Terbacil by Gavage in the Rabbit.* (Unpublished study, as cited in USEPA 1998).

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

Thelin, G.P. and L.P. Gianessi. 2000. *Method for Estimating Pesticide Use for County Areas of the Conterminous United States.* U.S. Geological Survey Open-File Report 00-250, 62 p. Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/ofr00250/ofr00250.pdf, accessed August 30, 2004.

Tomlin, C.D.S. (ed.) 1997. *The Pesticide Manual - World Compendium*, 11th Edition, British Crop Protection Council, Surrey, England. (As cited in HSDB, 2004).

United States Environmental Protection Agency (USEPA). 1990. *National Pesticide Survey: Survey Analytes.* EPA Report 570-9-90-NPS2. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS2.]

USEPA. 1992. *Pesticides in Ground Water Database: A Compilation of Monitoring Studies, 1971-1991. National Summary.* Office of Prevention, Pesticides and Toxic Substances. EPA Report 734-12-92-001. Available on the Internet at: http://nepis.epa.gov/pubtitleOPPTS.htm. [Search for document number 7341292001.]

USEPA. 1995a. *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1995b. Method 507 - Determination of Nitrogen and Phosphorus-Containing Pesticides in Water by Gas Chromatography with a Nitrogen-Phosphorus Detector. Revision 2.1. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1995c. Method 525.2 - Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry. Revision 2.0. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1998. Reregistration Eligibility Decision (RED)--Terbacil. EPA Report 738-R-97-011. Washington, DC: Office of Prevention, Pesticides and Toxic Substances. January 1998. Available on the Internet at: http://www.epa.gov/oppsrrd1/REDs/0039red.pdf.

USEPA. 2006a. TRI Explorer: Trends. Search for terbacil. Available on the Internet at: http://www.epa.gov/triexplorer/trends.htm. Accessed February 8, 2006. Last modified June 8, 2005.

USEPA. 2006b. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

United States Geological Survey (USGS). 2004. 1997 Pesticide Use Maps. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pesticide_use_maps_1997/. Accessed August 20, 2004.

Wazeter, F.X., R.H. Buller, and R.G. Geil. 1964. *Ninety-day Feeding Study in the Rat: IRDC No. 125-004.* (Unpublished study, as cited in USEPA, 1998).

Wazeter, F.X., R.H. Buller, and R.G. Geil. 1967a. *Two-Year Feeding Study in the Dog: IRDC No. 125-011*. (Unpublished study, as cited in USEPA, 1998).

Wazeter, F.X., R.H. Buller, and R.G. Geil. 1967b. *Three-Generation Reproduction Study in the Rat: IRDC No. 125-012*. (Unpublished study, as cited in USEPA, 1998).

Chapter 11: 1,1,2,2-Tetrachloroethane

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Executive Summary

1,1,2,2-Tetrachloroethane, a volatile organic compound (VOC), is not known to occur naturally in the environment. Prior to the 1980s, 1,1,2,2-tetrachloroethane was synthesized for use in the production of other chemicals, primarily chlorinated ethylenes. 1,1,2,2-Tetrachloroethane was also once used as a solvent to clean and degrease metals, in paint removers, varnishes, lacquers, and photographic films, and for oil/fat extraction. Commercial production of 1,1,2,2-tetrachloroethane in the U.S. ceased in the 1980s when other processes to generate chlorinated ethylenes were developed.

Volatilization from water or soil surfaces to the atmosphere appears to be the primary dissipation route for 1,1,2,2-tetrachloroethane. In subsurface soils and ground water, 1,1,2,2-tetrachloroethane is subject to biodegradation by soil organisms and/or chemical hydrolysis.

Recent studies by the National Toxicology Program (NTP) provide a detailed evaluation of the short-term and subchronic oral toxicity of 1,1,2,2-tetrachloroethane. In rats and mice exposed orally, the liver appears to be the primary target organ. The reference dose (RfD) of 10 µg/kg/day for 1,1,2,2-tetrachloroethane was derived from the benchmark dose level (BMDL) for a 1 standard deviation change in relative liver weight, a biomarker for liver toxicity. A 1,000-fold uncertainty factor was applied in the RfD determination.

A National Cancer Institute (NCI) bioassay of 1,1,2,2-tetrachloroethane found clear evidence of carcinogenicity in male and female B6C3F1 mice based on a dose-related statistically significant increase in liver tumors. There was equivocal evidence for carcinogenicity in Osborn Mendel rats. The Agency used the slope factor of 8.5×10^{-2} for the tumors in female mice to derive the health reference level (HRL) of 0.4 µg/L for use in the analysis of the occurrence data for 1,1,2,2-tetrachloroethane.

Individuals with preexisting liver and kidney damage would likely be more sensitive to 1,1,2,2-tetrachloroethane exposure than the general public. Low intake of antioxidant nutrients (e.g., Vitamin E, Vitamin C, and selenium) could be a predisposing factor for liver damage. Individuals with a genetically low capacity to metabolize dichloroacetic acid (the primary metabolite of 1,1,2,2-tetrachloroethane) may also be at elevated risk.

Production of 1,1,2,2-tetrachloroethane in the U.S. declined from approximately 440 million pounds in 1967 to an estimated 34 million pounds by 1974. Although U.S. commercial production ceased in the 1980s, 1,1,2,2-tetrachloroethane is still generated as a byproduct and/or intermediate in the production of other chemicals. Toxic Release Inventory (TRI) data indicate that environmental releases have generally declined from a high of about 175,000 pounds in 1988 to a low of 3,500 pounds in 2003. Most releases took the form of air emissions, though surface water discharges were also documented nearly every year.

The United States Geological Survey's (USGS's) Random Source Water Survey and Focused Source Water Survey, both conducted between 1999 and 2001, provide an indication of ambient occurrence of 1,1,2,2-tetrachloroethane. The USGS did not detect 1,1,2,2-tetrachloroethane in either survey using a reporting limit of 0.2 µg/L (a level that is less than the

1,1,2,2-tetrachloroethane HRL). In addition, USGS found no indication at all of 1,1,2,2-tetrachloroethane contamination above the detection limit of 0.026 µg/L in the focused survey. Additional sources of information on ambient occurrence include a USGS stormwater study and a USGS compilation of historical VOC monitoring data.

To determine the extent of 1,1,2,2-tetrachloroethane contamination in drinking water, EPA included 1,1,2,2-tetrachloroethane as an analyte in the Unregulated Contaminant Monitoring (UCM) Round 1 and UCM Round 2 surveys. EPA evaluated the UCM Round 1 Cross-Section and the UCM Round 2 Cross-Section data at levels greater than 0.2 µg/L (½ the HRL) and greater than 0.4 µg/L (the HRL). The minimum reporting levels (MRLs) for UCM Round 1 ranged from 0.1 to 10 µg/L and the MRLs for UCM Round 2 ranged from 0.1 to 2.5 µg/L for UCM Round 2. Because some of the reporting limits exceeded the thresholds of interest, the occurrence analyses may result in an underestimate of systems affected.

Analysis of UCM Round 1 Cross-Section data indicates that approximately 0.22 percent (or 44) of the 20,407 public water systems (PWSs) sampled had detections of 1,1,2,2-tetrachloroethane at levels greater than 0.20 µg/L (½ the HRL), affecting approximately 1.69 percent of the population served (or 1.6 million of 95 million). The UCM Round 1 Cross-Section data indicate that approximately 0.20 percent (or 41) of the 20,407 PWSs sampled had detections of 1,1,2,2-tetrachloroethane at levels greater than 0.4 µg/L (the HRL), affecting approximately 1.63 percent of the population served (or 1.5 million of 95 million). The 99th percentile of all detects was 112 µg/L and the maximum reported value was 200 µg/L.

Analysis of the UCM Round 2 Cross-Section data indicate that approximately 0.07 percent (or 18) of the 24,800 PWSs sampled had detections of 1,1,2,2-tetrachloroethane at levels greater than 0.2 µg/L (½ the HRL), affecting approximately 0.51 percent of the population served (or 362,000 of 71 million). The UCM Round 2 Cross-Section data indicate that approximately the same percentage and number of the PWSs sampled (0.07 percent or 17 of the 24,800) had detections of 1,1,2,2-tetrachloroethane at levels greater than 0.4 µg/L (the HRL), affecting approximately 0.08 percent of the population served (or 56,000 of 71 million). The 99th percentile of all detects was 2 µg/L and the maximum reported value was 2 µg/L.

The Agency has made a preliminary determination not to regulate 1,1,2,2-tetrachloroethane with a national primary drinking water regulation (NPDWR). Because 1,1,2,2-tetrachloroethane appears to occur infrequently at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction.

EPA recognizes that 1,1,2,2-tetrachloroethane is listed as a likely human carcinogen. For this reason, the Agency encourages those States with public water systems that may have 1,1,2,2-tetrachloroethane above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory document for 1,1,2,2-tetrachloroethane to provide more recent health information.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

Contents

Exhibits

Abbreviations

BMDL	Benchmark Dose Level
CAS	Chemical Abstracts Service
CCL	Contaminant Candidate List
CCL 2	Second Contaminant Candidate List
ELCD	Electrolytic Conductivity Detector
GAC	Granular Activated Carbon
GC	Gas Chromatography
HRL	Health Reference Level
LOAEL	Lowest Observed Adverse Effect Level
MDL	Method Detection Limit
MRL	Minimum Reporting Level
MS	Mass Spectrometry
NAWQA	National Water Quality Assessment
NOAEL	No Observed Adverse Effect Level
NPDES	National Pollutant Discharge Elimination System
NPDWR	National Primary Drinking Water Regulation
NTP	National Toxicology Program
PCE	Tetrachloroethylene
PID	Photoionization Detector
PWS	Public Water System
RfD	Reference Dose
RL	Reporting Limit
TCE	Trichloroethylene
TRI	Toxics Release Inventory
UCM	Unregulated Contaminant Monitoring
USGS	United States Geological Survey
VOC	Volatile Organic Compound

11 1,1,2,2-Tetrachloroethane

11.1 Definition

1,1,2,2-Tetrachloroethane is a halogenated volatile organic compound (VOC) used in chemical synthesis. It is also given the following chemical names: acetosol, acetylene tetrachloride, symmetrical-tetrachloroethane, sym-tetrachloroethane, 1,1-dichloro-2,2-dichloroethane, and tetrachloroethane. 1,1,2,2-Tetrachloroethane goes by three registered trade names: Bonoform, Cellon, and Westron. The Chemical Abstracts Service (CAS) registry number for 1,1,2,2-tetrachloroethane is 79-34-5.

11.1.1 Properties and Sources

1,1,2,2-Tetrachloroethane is not known to occur naturally (IARC, 1979 as cited in ATSDR, 1996). At room temperature it is a dense, colorless liquid with a pungent, sweet, suffocating, chloroform-like smell. It is produced by the catalytic addition of chlorine to acetylene or through the direct chlorination or oxychlorination of ethylene (IARC, 1979; Archer, 1979 both as cited in ATSDR, 1996). Prior to the 1980s, the Specialty Materials Division of Eagle-Picher Industries synthesized this chemical for use in the production of other chemicals, primarily chlorinated ethylenes, as well as use as a solvent. Commercial production was discontinued in the 1980s when other methods to generate chlorinated ethylenes were discovered. The present use of 1,1,2,2-tetrachloroethane appears to be mostly as a chemical intermediate (ATSDR, 1996), although it is also produced as a by-product in the synthesis of other chlorinated hydrocarbons (Gerhartz, 1985 as cited in HSDB, 2004). Some physical and chemical properties of this VOC are summarized in Exhibit 11-1.

Exhibit 11-1: Physical and Chemical Properties of 1,1,2,2-Tetrachloroethane

Identification	
CAS number	79-34-5
Molecular Formula	$C_2H_2Cl_4$
Physical and Chemical Properties	
Boiling Point	146.5 °C at 760 mm Hg [1]
Melting Point	- 43.8 ° C [1]
Molecular Weight	167.85 g/mol [1]
Log K_{oc}	2.78 [2]
Log K_{ow}	2.39 [3]
Water Solubility	2,962 mg/L at 25 °C [4]
Vapor Pressure	6.1 mm Hg at 25 ° C [5]
Henry's Law Constant	4.55×10^{-4} atm-m^3/mole at 25 ° C [5] 0.012 mol/mol (dimensionless), predicted [6] 0.016 mol/mol (dimensionless), from literature [6]
Freundlich Isotherm Constant (K)	823 (µg/g)(L/µg)$^{1/n}$ [7]

[1] *Lide, 1995 as cited in HSDB, 2004*

[2] *ASTER, 1995 as cited in ATSDR, 1996*

[3] *Hansch et al., 1995 as cited in HSDB, 2004*

[4] *Horvath, 1982 as cited in HSDB, 2004*

[5] *Howard, 1990*

[6] *Speth et al., 2001*

[7] *Speth and Adams, 1993 (as cited in Speth et al., 2001)*

11.1.2 Environmental Fate and Behavior

The evaporation of 1,1,2,2-tetrachloroethane from soil surfaces is expected to be fairly rapid (HSDB, 2004). In silt loam, 1,1,2,2-tetrachloroethane has been found to be highly mobile, suggesting a potential for leaching to ground water (Howard, 1990). Experiments simulating degradation reactions under landfill conditions found 1,1,2,2-tetrachloroethane to transform to a number of products, including 1,1,2-trichloroethane, trichloroethene, 1,1-dichloroethene, and vinyl chloride (Hallen *et al.*, 1986 as cited in ATSDR, 1996).

A large percentage of 1,1,2,2-tetrachloroethane released to water will evaporate with a half-life of days to weeks depending on the water body (Howard, 1990). The remaining portion will degrade through hydrolysis. In ground water, 1,1,2,2-tetrachloroethane will degrade through anaerobic biodegradation or hydrolysis. Hydrolysis is pH-dependant - degradation will be faster under basic to neutral conditions. At a neutral pH, 1,1,2,2-tetrachloroethane hydrolysis half-lives range from 29 to 102 days (Haag and Mill, 1988; Cooper *et al.*, 1987 both as cited in

ATSDR, 1996). Trichloroethylene is the major product of 1,1,2,2-tetrachloroethane hydrolysis, while biodegradation is reported to produce 1,1,2-trichloroethane (Bouwer and McCarty, 1983 as cited in Howard, 1990). Adsorption of 1,1,2,2-tetrachloroethane to stream sediments and bioconcentration in fish is expected to be minimal (Howard, 1990).

As a highly volatile chemical with slow biodegradation in soil and water, most 1,1,2,2-tetrachloroethane releases to any medium will eventually enter the atmosphere. In the atmosphere, 1,1,2,2-tetrachloroethane will disperse and eventually degrade by reaction with photochemically produced hydroxyl radicals. The half-life for this process has been theoretically estimated to be 53 days (Atkinson, 1987 as cited in ATSDR, 1996). Older experimental data suggest that 1,1,2,2-tetrachlorethane may have a significantly longer residence time in the atmosphere, with a half-life of two years (Singh *et al.*, 1981 as cited in HSDB, 2004). Due to potentially long residence times in the atmosphere, a small percentage (~1 percent) of 1,1,2,2-tetrachloroethane is predicted to escape to the stratosphere where it will rapidly degrade through photodissociation (Howard, 1990).

11.2 Health Effects

Data on the toxicity of 1,1,2,2-tetrachloroethane in humans are limited, consisting of one experimental inhalation study, a few case reports of suicidal or accidental ingestion, and dated occupational studies. In most cases, there was no quantification of the exposure. Respiratory and mucosal effects, eye irritation, nausea, vomiting, and dizziness were reported by human volunteers exposed to 1,1,2,2-tetrachloroethane vapors under controlled chamber conditions (Lehmann and Schmidt-Kehl, 1936 as cited in ATSDR, 1996 and USEPA, 1989). Effects from non-lethal occupational exposures included gastric distress (i.e., pain, nausea, vomiting), headache, loss of appetite, an enlarged liver, and cirrhosis (Jeney *et al.*, 1957 as cited in USEPA 1989; Lobo-Mendonca, 1963 as cited in ATSDR, 1996 and USEPA, 1989; Minot and Smith 1921 as cited in ATSDR, 1996).

There have been a variety of animal studies in rats and mice using both the inhalation and oral exposure routes. Recent studies by the National Toxicology Program (NTP, 2004) provide a detailed evaluation of the short-term and subchronic oral toxicity of 1,1,2,2-tetrachloroethane and confirm many of the observations from earlier studies. In rats and mice exposed orally, the liver appears to be the primary target organ. The reference dose (RfD) (10 µg/kg/day) for 1,1,2,2-tetrachloroethane was derived from the benchmark dose level ($BMDL_{10}$) for a 1 standard deviation change in relative liver weight, a biomarker for liver toxicity. A 1,000-fold uncertainty factor was applied in the RfD determination.

A National Cancer Institute (1978 as cited in ATSDR, 1996) bioassay of 1,1,2,2-tetrachloroethane found clear evidence of carcinogenicity in male and female B6C3F1 mice based on a dose-related statistically significant increase in liver tumors. There was equivocal evidence for carcinogenicity in Osborn Mendel rats because of the occurrence of a small number of rare-for-the species neoplastic and preneoplastic lesions in the livers of the high dose animals. The Agency used the slope factor of 8.5×10^{-2} for the tumors in female mice to derive the health reference level (HRL) of 0.4 µg/L for use in the analysis of the occurrence data for 1,1,2,2-tetrachloroethane.

Information on the reproductive effects of 1,1,2,2-tetrachloroethane is limited. There is a single one-generation inhalation study that does not follow a standard methodology and examined a small number of rats (five females and seven males) exposed via inhalation to one dose (13.3 mg/m^3). There were no statistically significant differences in the percentage of females having offspring, number of pups per litter, average birth weight, sex ratio, or post natal offspring mortality (Schmidt *et al.*, 1972). Effects on sperm in male rats were seen after oral (27 mg/kg/day; NTP, 2004) and inhalation (13 mg/m^3; Schmidt *et al.*, 1972) exposures. Similar effects were seen in mice but at higher doses. Fetal toxicity did not occur in the absence of maternal toxicity.

Developmental range-finding studies conducted for NTP (1991a, 1991b) found that 1,1,2,2-tetrachloroethane was toxic to the dams and pups of Sprague Dawley rats and CD-1 Swiss mice. Rats were more sensitive than mice. The "no-observed-adverse-effect level" (NOAEL) in the rats for both maternal toxicity and associated fetal toxicity was 34 mg/kg/day with a "lowest-observed-adverse-effect level" (LOAEL) of 98 mg/kg/day. In mice, the NOAEL was 987 mg/kg/day and the LOAEL was 2,120 mg/kg/day.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. Individuals with preexisting liver and kidney damage would likely be sensitive to 1,1,2,2-tetrachloroethane exposure. Low intake of antioxidant nutrients (e.g., Vitamin E, Vitamin C, and selenium) could be a predisposing factor for liver damage. In addition, individuals with a genetically low capacity to metabolize dichloroacetic acid (the primary metabolite of 1,1,2,2-tetrachloroethane) may be at greater risk than the general population as a result of 1,1,2,2-tetrachloroethane exposure.

11.3 Occurrence and Exposure

11.3.1 Use and Environmental Release

Prior to the 1980s, 1,1,2,2-tetrachloroethane was commonly used in the production of other chemicals, primarily trichloroethylene (TCE), tetrachloroethylene (PCE), and 1,2-dichloroethylene (Archer, 1979 as cited in ATSDR, 1996). It was also used as a metal degreaser, an extractant for oils and fats, and a component of paint removers, varnishes and lacquers, and photographic films (Hawley, 1981 as cited in ATSDR, 1996). At one time the compound was also used as an insecticide, fumigant, weedkiller, and insect repellant, but it is not currently registered in the United States for such uses. Approximately 440 million pounds of 1,1,2,2-tetrachloroethane were produced in 1967 (Konietzko, 1984 as cited in ATSDR, 1996). Production fell to 34 million pounds in 1974, and production for commercial uses ceased in the United States by the late 1980s. Imports are also thought to be minimal (ATSDR, 1996).

Although 1,1,2,2-tetrachloroethane is no longer generated as an end product, it is still generated as an intermediate product and/or by-product in the manufacturing of other synthetic chemicals, including trichloroethylene, 1,1,2-trichloroethane, 1,2-dichloroethene, tetrachloroethylene, vinyl chloride, ethylene dichloride, and 1,1,1-trichloroethane. It can occur as a trace contaminant in these and other manufactured chemicals, and in the waste stream of facilities that produce them. ATSDR (1996) lists 15 facilities that produce 1,1,2,2-

tetrachloroethane as a by-product or use it as an intermediate product. (Note: The list is likely not exhaustive.)

1,1,2,2-Tetrachloroethane is listed as a Toxics Release Inventory (TRI) chemical. For a discussion of the nature and limitations of TRI data, see Chapter 2.

TRI data for 1,1,2,2-tetrachloroethane (see Exhibit 11-2) are reported for the years 1988 to 2003 (USEPA, 2006a). Air emissions constitute most of the on-site releases. Reported air releases peaked in 1991 and then generally declined. Surface water discharges ranged in the thousands of pounds until the mid-1990s, and then dropped off significantly until a sharp increase in 2002. There is no detectable pattern in on-site underground injections or releases to land. Reported off-site releases were most significant in the first year of reporting, and then generally declined, with an aberrant peak in 1998. These TRI data for 1,1,2,2-tetrachloroethane were reported from 20 States (AR, CA, CO, CT, FL, KS, KY, LA, MI, MO, NC, NE, NJ, NY, OH, PA, SC, TN, TX, VA), but no more than 11 States reported in a given year. Louisiana and Texas were the only States to report releases every year.

Exhibit 11-2: Environmental Releases (in pounds) of 1,1,2,2-Tetrachloroethane in the United States, 1988-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1988	43,865	1,903	0	29	128,750	174,547
1989	35,611	5,429	283	18	15,209	56,550
1990	44,796	3,529	80	495	771	49,671
1991	64,251	2,113	0	0	262	66,626
1992	48,899	5,164	0	0	273	54,336
1993	28,203	2,930	0	1	80	31,214
1994	12,484	1,517	26	0	52	14,079
1995	8,275	2,222	0	0	7	10,504
1996	15,488	130	0	0	7	15,625
1997	13,614	0	0	0	511	14,125
1998	7,299	269	5	0	6,503	14,076
1999	5,202	1	0	15	30	5,248
2000	4,461	13	5	0	631	5,110
2001	3,462	56	0	961	941	5,420
2002	7,879	1,464	0	1	108	9,452
2003	2,729	466	0	66	259	3,520

Source: USEPA, 2006a

11.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are sources of drinking water. Data on the occurrence of 1,1,2,2-tetrachloroethane in ambient surface and ground water are available from the National Water Quality Assessment (NAWQA) program of the United States Geological Survey (USGS). For further details on this program, see the discussion of NAWQA in Chapter 2. NAWQA data have been analyzed independently by USGS and EPA. USGS has also collected data on 1,1,2,2-tetrachloroethane occurrence in a review of stormwater studies.

NAWQA VOC National Synthesis

Random and Focused VOC Surveys

Using data collected from the NAWQA Study Units and other sources, USGS and collaborating institutions have recently completed a national synthesis assessment of VOC occurrence in the nation's drinking water supply. The assessment included a random survey (1999-2000) of VOC occurrence in ground and surface water resources used by geographically representative community water systems in different size categories (Grady, 2003) and a focused survey (1999-2001) of VOC occurrence patterns, including seasonal variability, in source waters considered particularly susceptible to MTBE contamination (Delzer and Ivahnenko, 2003). 1,1,2,2-Tetrachloroethane was included as an analyte in both surveys, with a reporting limit of 0.2 µg/L (Ivahnenko *et al.*, 2001).

The national random survey and focused survey both found no detections of 1,1,2,2-tetrachloroethane at the reporting level of 0.2 µg/L (Grady, 2003; Delzer and Ivahnenko, 2003). In addition, the focused survey provided results for 1,1,2,2-tetrachloroethane below the reporting level. At levels as low as the method detection limit (0.026 µg/L), no detections of 1,1,2,2-tetrachloroethane were found (Delzer and Ivahnenko, 2003).

Compilation of Historical VOC Monitoring Data

USGS assessed VOC occurrence in untreated ambient ground water samples collected between 1985 and 1995 by local, State, and federal agencies (Squillace *et al.*, 1999). The samples represented both urban and rural areas, and both drinking water and non-drinking water wells.

Multiple investigators collected 1,1,2,2-tetrachloroethane samples from 204 urban wells and 1,267 rural wells. At a reporting level of 0.2 µg/L, there were no detections of 1,1,2,2-tetrachloroethane.

EPA Summary Analysis of NAWQA Data

Whereas the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination, including 1,1,2,2-tetrachloroethane. Detection frequencies were simply computed as the percentage of samples and sites with detections (i.e., with at least one result equal to or greater than the reporting limit). Note that reporting limits were not uniform. Sample detections can be biased by frequent sampling in areas with high (or low) occurrence. Calculating the percentage of sites with detections can reduce this bias. For more details on the data set and the EPA analysis, see Chapter 2.

The results of the EPA analysis are presented in Exhibit 11-3. Overall, 1,1,2,2-tetrachloroethane was detected in 0.07% of samples and at 0.07% of sites. 1,1,2,2-Tetrachloroethane was detected more frequently in surface water but at higher concentrations (maximum of 0.38 µg/L) in ground water.

Exhibit 11-3: EPA Summary Analysis of 1,1,2,2-Tetrachloroethane Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	1,408	0.21%	190	1.05%	0.02	0.08	0.20	0.20	0.20
ground water	4,544	0.02%	4,127	0.02%	0.38	0.38	0.38	0.38	0.38
all sites	5,952	0.07%	4,317	0.07%	0.02	0.14	0.38	0.38	0.38

[1] *RLs (Reporting Limits) for 1,1,2,2-tetrachloroethane varied but did not exceed 0.2 µg/L. For more information, see Chapter 2. Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible.*

USGS Stormwater Studies

For the National Highway Runoff Data and Methodology Synthesis, USGS conducted a review of 44 highway and urban runoff studies implemented since 1970 (Lopes and Dionne, 1998). 1,1,2,2-Tetrachloroethane results are reported in four of these studies. For background information on this review, see Chapter 2.

Three of the studies were stormwater studies conducted in major metropolitan areas in connection with National Pollutant Discharge Elimination System (NPDES) permitting. In metropolitan Phoenix (Maricopa County), USGS collected 35 samples from 5 drainage basins and the City of Phoenix collected an additional 26 samples from 7 sites (Lopes *et al.*, 1995). In Colorado Springs, 35 samples were collected from 5 sites (von Guerard and Weiss, 1995). In Dallas-Fort Worth, 182 samples were collected from 26 stormwater drainage basins (Baldys *et al.*, 1998). The reporting limits were 0.2 µg/L in Phoenix and Colorado Springs, and they ranged from 0.2 to 10 µg/L in Dallas-Fort Worth. Not all samples were monitored for every contaminant. These three studies found no detections of 1,1,2,2-tetrachloroethane above the reporting limits.

The fourth study analyzed 86 urban runoff samples from 15 U.S. cities, collected between 1979 and 1982 in connection with the National Urban Runoff Program (Cole *et al.*, 1984). 1,1,2,2-Tetrachloroethane was detected in 2 percent of samples, in concentrations ranging from 2 µg/L to 3 µg/L. All detections were from Long Island, New York. A detection limit was not reported.

11.3.3 Drinking Water Occurrence

Nationally representative data on 1,1,2,2-tetrachloroethane occurrence in drinking water were collected by large and small public water systems under EPA's Unregulated Contaminant Monitoring (UCM) program (1987-1999).

UCM Program, Rounds 1 and 2

Round 1 of the UCM lasted from 1988 to 1992, and Round 2 lasted from 1993 to 1999. A geographical cross-section of States with the most complete and reliable data was chosen to provide a roughly representative picture of national occurrence in each round. For more details on the UCM program, see Chapter 2 and USEPA (2006b).

Exhibits 11-4 and 11-5 show the results from the Round 1 and Round 2 cross-sections. Results from all States, including those with incomplete and less reliable data, are also presented for the sake of comparison. Results are analyzed at the level of simple detections (at or above the minimum reporting level, or ≥ MRL), exceedances of the health reference level (> HRL, or > 0.4 µg/L), and exceedances of one half the value of the HRL (> ½ HRL, or > 0.2 µg/L). MRLs for 1,1,2,2-tetrachloroethane were not uniform. They varied from 0.01 µg/L to 10 µg/L in the first round, and from 0.01 µg/L to 2.5 µg/L in the second round. The modal (most common) MRL in both rounds was 0.5 µg/L. Because the MRL was often higher than the HRL and ½ HRL, it is likely that the sampling failed to capture some HRL and ½ HRL exceedances at the participating systems, and that the HRL and ½ HRL analyses underestimate actual 1,1,2,2-tetrachloroethane occurrence. However, all MRLs fell within (or below) the risk range of 10^{-6} to 10^{-4} used by EPA to evaluate carcinogens (see Section 2.1.1).

In Round 1 cross-section States, 1,1,2,2-tetrachloroethane was detected at approximately 0.45% of public water systems (PWSs), affecting 1.86% of the population served, equivalent to approximately 4.0 million people nationally. Exceedances of one-half the value of the HRL were found at 0.22% of PWSs, affecting 1.69% of the population served, equivalent to approximately 3.6 million people nationally. HRL exceedances were found at 0.20% of PWSs, affecting 1.63% of the population served, equivalent to approximately 3.5 million people nationally.

When all Round 1 results are included in the analysis, including results from States with incomplete or less reliable data, 1,1,2,2-tetrachloroethane detection frequencies appear to be slightly higher than the cross-section data indicate. Detections affect 0.48% of PWSs and 2.16% of the population served; exceedances of the ½ HRL benchmark affect 0.26% of PWSs and 1.99% of the population served; and HRL exceedances affect 0.24% of PWSs and 1.90% of the population served.

In Round 2 cross-section States, 1,1,2,2-tetrachloroethane was detected at 0.08% of PWSs, affecting 2.61% of the population served, equivalent to approximately 5.6 million people nationally. The ½ HRL benchmark was exceeded in 0.07% of PWSs (18 of 24,800), affecting 0.51% of the population served, equivalent to approximately 1.1 million people nationally. The HRL benchmark was exceeded in 0.07% of PWSs (17 of 24,800—one fewer than the ½ HRL benchmark), affecting 0.08% of the population served, equivalent to approximately 0.2 million people nationally. Round 2 generally shows lower occurrence of 1,1,2,2-tetrachloroethane than Round 1. One apparently contradictory indicator, the strikingly high proportion of the population served by PWSs with detections in Round 2, is due to the unusually large size of one of the relatively few contaminated surface water systems.

Including Round 2 results from all reporting States in the analysis does not change the picture of 1,1,2,2-tetrachloroethane occurrence significantly. Detections affect 0.08% of PWSs and 2.23% of the population served; ½ HRL exceedances affect 0.07% of PWSs and 0.44% of the population served; and HRL exceedances affect 0.06% of PWSs and 0.08% of the population served.

Exhibit 11-4: Summary UCM Occurrence Statistics for 1,1,2,2-Tetrachloroethane (Round 1)

Frequency Factors	24-State Cross-Section[1]		All Reporting States[2]		National System & Population Numbers[3]	
Total Number of Samples	67,688		70,784		--	
Percent of Samples with Detections	0.16%		0.16%		--	
99th Percentile Concentration (all samples)	< MRL		< MRL		--	
Health Reference Level (HRL)	0.4 µg/L		0.4 µg/L		--	
Minimum Reporting Level (MRL) - Range - (modal value)[4]	0.01 - 10 µg/L (0.5 µg/L)		0.01 - 10 µg/L (0.5 µg/L)		--	
Maximum Concentration of Detections	200 µg/L		200 µg/L		--	
99th Percentile Concentration of Detections	112 µg/L		112 µg/L		--	
Median Concentration of Detections	0.5 µg/L		0.5 µg/L		--	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	20,407 18,693 1,867		20,899 19,054 2,019		65,030 59,440 5,590	
Total Population Population of GW PWSs Population of SW PWSs	94,710,065 55,763,644 43,763,942		98,334,686 57,663,608 45,776,159		213,008,182 85,681,696 127,326,486	
Occurrence by System	**Number**	**Percentage**	**Number**	**Percentage**	**National Extrapolation[5]**	
					Cross-Section	**All States**
PWSs with detections (≥ MRL) Range across States GW PWSs with detections SW PWSs with detections	91 0 - 39 72 19	0.45% 0 - 11.64% 0.39% 1.02%	101 0 - 39 80 21	0.48% 0 - 100% 0.42% 1.04%	290 N/A 229 57	314 N/A 250 58
PWSs > 1/2 HRL Range across States GW PWSs > 1/2 HRL SW PWSs > 1/2 HRL	44 0 - 11 33 11	0.22% 0 - 2.76% 0.18% 0.59%	54 0 - 11 41 13	0.26% 0 - 100% 0.22% 0.64%	140 N/A 105 33	168 N/A 128 36
PWSs > HRL Range across States GW PWSs > HRL SW PWSs > HRL	41 0 - 11 32 9	0.20% 0 - 2.76% 0.17% 0.48%	50 0 - 11 39 11	0.24% 0 - 100% 0.20% 0.54%	131 N/A 102 27	156 N/A 122 30
Occurrence by Population Served						
Population served by PWSs with detections Range across States Pop. Served by GW PWSs with detections Pop. Served by SW PWSs with detections	1,762,198 0 - 616,019 1,017,630 744,568	1.86% 0 - 25.48% 1.82% 1.70%	2,119,844 0 - 616,019 1,365,976 753,868	2.16% 0 - 100% 2.37% 1.65%	3,963,000 N/A 1,564,000 2,166,000	4,592,000 N/A 2,030,000 2,097,000
Population served by PWSs > 1/2 HRL Range across States Pop. Served by GW PWSs > 1/2 HRL Pop. Served by SW PWSs > 1/2 HRL	1,597,140 0 - 616,019 864,770 732,370	1.69% 0 - 25.48% 1.55% 1.67%	1,954,786 0 - 616,019 1,213,116 741,670	1.99% 0 - 100% 2.10% 1.62%	3,592,000 N/A 1,329,000 2,131,000	4,234,000 N/A 1,803,000 2,063,000
Population served by PWSs > HRL Range across States Pop. Served by GW PWSs > HRL Pop. Served by SW PWSs > HRL	1,543,647 0 - 616,019 851,641 692,006	1.63% 0 - 25.48% 1.53% 1.58%	1,868,493 0 - 616,019 1,167,187 701,306	1.90% 0 - 100% 2.02% 1.53%	3,472,000 N/A 1,309,000 2,013,000	4,047,000 N/A 1,734,000 1,951,000

1. Summary Results based on 24-State Cross-Section, UCM Round 1 data.
2. Summary Results based on All Reporting States, UCM Round 1 data.
3. Total PWS and population numbers are from EPA March 2000 Water Industry Baseline Handbook, 2nd Edition.
4. Because several different analytical methods were used, MRLs were not uniform. The modal value is the most common MRL.
5. National extrapolations are generated by multiplying the system/population percentages and the national Baseline Handbook system/population numbers.

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with Detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with Detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-Because some systems were counted as both ground water and surface water systems and others could not be classified, GW and SW figures might not add up to totals.
-Due to differences between the ratios of GW and SW systems with monitoring results and the national ratio, extrapolated GW and SW figures might not add up to extrapolated totals.
-Due to MRL variability, it is likely that the sampling failed to capture some ½ HRL and HRL exceedances at the participating systems, and the ½ HRL and HRL analyses underestimate actual contaminant occurrence.
-The HRL used in this analysis is a draft value for working review only.

Exhibit 11-5: Summary UCM Occurrence Statistics for 1,1,2,2-Tetrachloroethane (Round 2)

Frequency Factors	20-State Cross-Section[1]		All Reporting States[2]		National System & Population Numbers[3]	
Total Number of Samples	98,911		112,480		--	
Percent of Samples with Detections	0.02%		0.03%		--	
99th Percentile Concentration (all samples)	< MRL		< MRL		--	
Health Reference Level (HRL)	0.4 µg/L		0.4 µg/L		--	
Minimum Reporting Level (MRL) - Range - (modal value)[4]	0.1 - 2.5 µg/L (0.5 µg/L)		0.1 - 2.5 µg/L (0.5 µg/L)		--	
Maximum Concentration of Detections	2 µg/L		3.9 µg/L		--	
99th Percentile Concentration of Detections	2 µg/L		3.9 µg/L		--	
Median Concentration of Detections	0.5 µg/L		0.5 µg/L		--	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	24,800 22,106 2,694		28,209 25,152 3,057		65,030 59,440 5,590	
Total Population Population of GW PWSs Population of SW PWSs	71,294,263 25,978,359 45,315,904		84,692,367 31,069,576 53,622,791		213,008,182 85,681,696 127,326,486	
Occurrence by System	**Number**	**Percentage**	**Number**	**Percentage**	**National Extrapolation[5]**	
					Cross-Section	**All States**
PWSs with detections (≥ MRL) Range across States GW PWSs with detections SW PWSs with detections	19 0 - 9 11 8	0.08% 0 - 0.50% 0.05% 0.30%	22 0 - 9 13 9	0.08% 0 - 3.49% 0.05% 0.29%	50 N/A 30 17	51 N/A 31 16
PWSs > 1/2 HRL Range across States GW PWSs > 1/2 HRL SW PWSs > 1/2 HRL	18 0 - 9 11 7	0.07% 0 - 0.50% 0.05% 0.26%	19 0 - 9 12 7	0.07% 0 - 1.16% 0.05% 0.23%	47 N/A 30 15	44 N/A 28 13
PWSs > HRL Range across States GW PWSs > HRL SW PWSs > HRL	17 0 - 9 11 6	0.07% 0 - 0.50% 0.05% 0.22%	18 0 - 9 12 6	0.06% 0 - 1.16% 0.05% 0.20%	45 N/A 30 12	41 N/A 28 11
Occurrence by Population Served						
Population served by PWSs with detections Range across States Pop. Served by GW PWSs with detections Pop. Served by SW PWSs with detections	1,862,105 0 - 1,500,000 24,115 1,837,990	2.61% 0 - 29.92% 0.09% 4.06%	1,892,850 0 - 1,500,000 51,543 1,841,307	2.23% 0 - 29.92% 0.17% 3.43%	5,563,000 N/A 80,000 5,164,000	4,761,000 N/A 142,000 4,372,000
Population served by PWSs > 1/2 HRL Range across States Pop. Served by GW PWSs > 1/2 HRL Pop. Served by SW PWSs > 1/2 HRL	362,105 0 - 306,000 24,115 337,990	0.51% 0 - 7.12% 0.09% 0.75%	371,980 0 - 306,000 33,990 337,990	0.44% 0 - 7.12% 0.11% 0.63%	1,082,000 N/A 80,000 950,000	936,000 N/A 94,000 803,000
Population served by PWSs > HRL Range across States Pop. Served by GW PWSs > HRL Pop. Served by SW PWSs > HRL	56,105 0 - 26,550 24,115 31,990	0.08% 0 - 0.54% 0.09% 0.07%	65,980 0 - 26,550 33,990 31,990	0.08% 0 - 0.54% 0.11% 0.06%	168,000 N/A 80,000 90,000	166,000 N/A 94,000 76,000

1. Summary Results based on 20-State Cross-Section, UCM Round 2 data.
2. Summary Results based on All Reporting States, UCM Round 2 data.
3. Total PWS and population numbers are from EPA March 2000 Water Industry Baseline Handbook, 2nd Edition.
4. Because several different analytical methods were used, MRLs were not uniform. The modal value is the most common MRL.
5. National extrapolations are generated by multiplying the system/population percentages and the national Baseline Handbook system/population numbers.

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with Detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with Detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-Due to differences between the ratios of GW and SW systems with monitoring results and the national ratio, extrapolated GW and SW figures might not add up to extrapolated totals.
-Due to MRL variability, it is likely that the sampling failed to capture some ½ HRL and HRL exceedances at the participating systems, and the ½ HRL and HRL analyses underestimate actual contaminant occurrence.
-The HRL used in this analysis is a draft value for working review only.

Each of the following maps focuses on a somewhat different aspect of the geographical distribution of 1,1,2,2-tetrachloroethane occurrence. Exhibit 11-6 identifies all States with at least one PWS with a detection of 1,1,2,2-tetrachloroethane in Round 1 or Round 2. All States are included in this analysis, including both cross-section States with reliable data and non-cross-section States with less reliable data, in order to provide the broadest assessment of possible 1,1,2,2-tetrachloroethane occurrence. Exhibit 11-7 presents the same information (identifying States with detections, regardless of whether they were included in the cross-sections) separately for Round 1 (1988-1992) and Round 2 (1993-1999), to reveal temporal trends.

Exhibit 11-8 illustrates the geographic distribution of States with different detection frequencies (percentage of PWSs with at least one detection), and Exhibit 11-9 illustrates the geographic distribution of different HRL exceedance frequencies (percentage of PWSs with at least one HRL exceedance). Only cross-section States, which have the most complete and reliable occurrence data, are included in these two analyses. In each exhibit, Round 1 data are presented in the upper map and Round 2 data are presented in the lower map to reveal temporal trends.

In each map, two color categories represent States with no data. Those in white do not belong to the relevant Round or cross-section, and those in the lightest category of shading were included in the Round or cross-section but have no data for 1,1,2,2-tetrachloroethane. The darker shades are used to differentiate occurrence findings in States with 1,1,2,2-tetrachloroethane data.

The large number of Northeastern and Great Lakes States reporting at least one detection, especially in Round 1, suggests a possible regional problem. However, States with detections are distributed from the east to the west coast, and from the Canadian to the Mexican borders. Even the States with the highest proportion of PWSs with detections are generally distributed across the United States.

Exhibit 11-6: Geographic Distribution of 1,1,2,2-Tetrachloroethane Detections in Both Cross-Section and Non-Cross-Section States (Combined UCM Rounds 1 and 2)

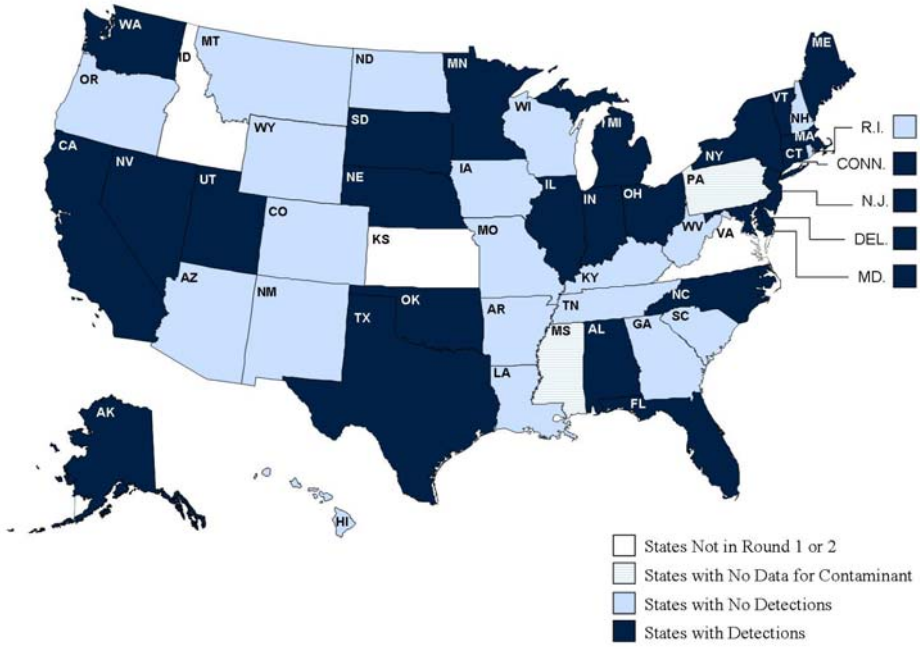
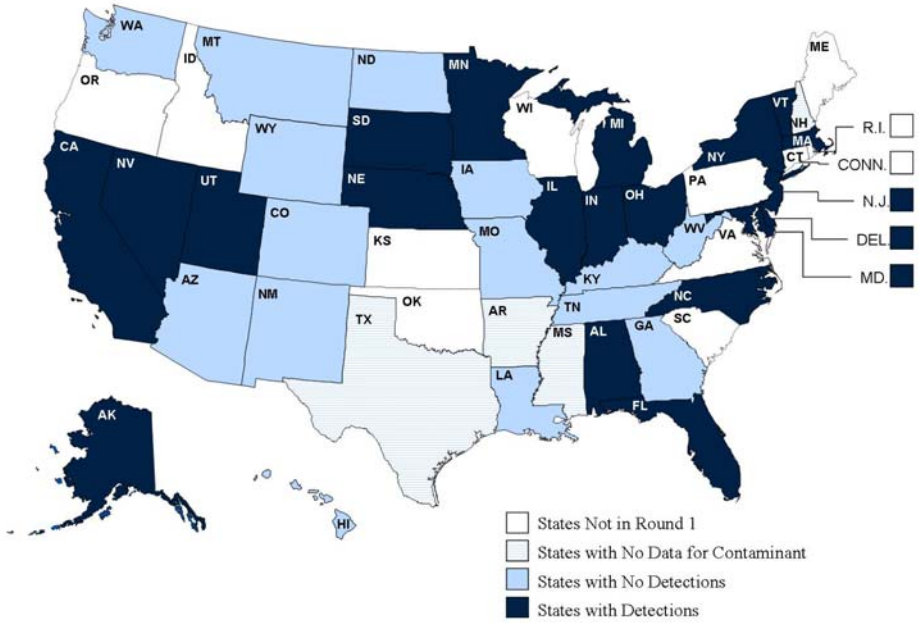


Exhibit 11-7: Geographic Distribution of 1,1,2,2-Tetrachloroethane Detections in Both Cross-Section and Non-Cross-Section States (Above: UCM Round 1; Below: UCM Round 2)



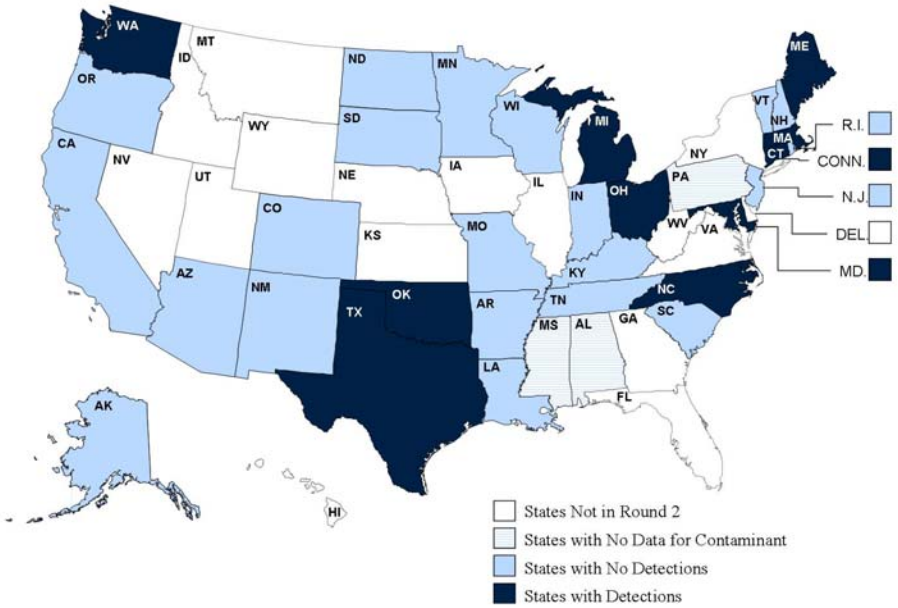
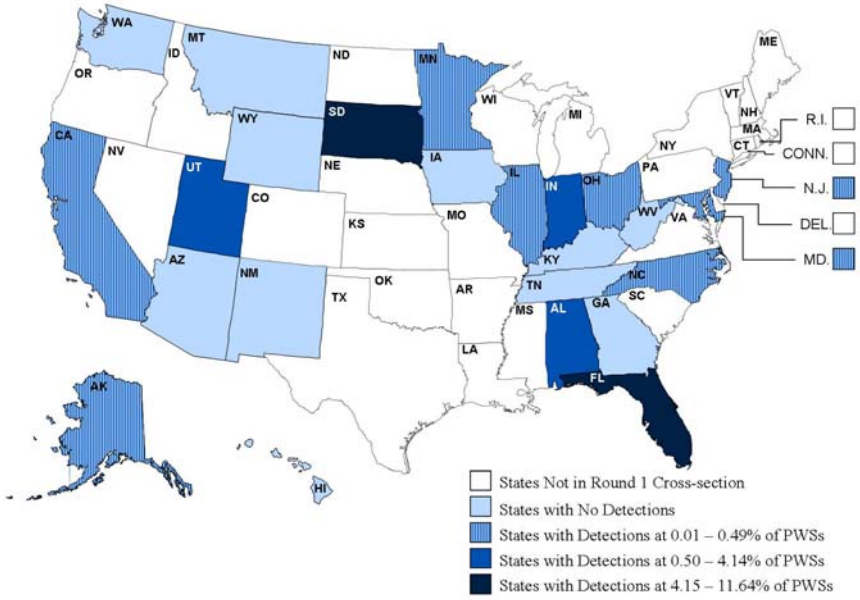


Exhibit 11-8: Geographic Distribution of 1,1,2,2-Tetrachloroethane Detection Frequencies in Cross-Section States (Above: UCM Round 1; Below: UCM Round 2)



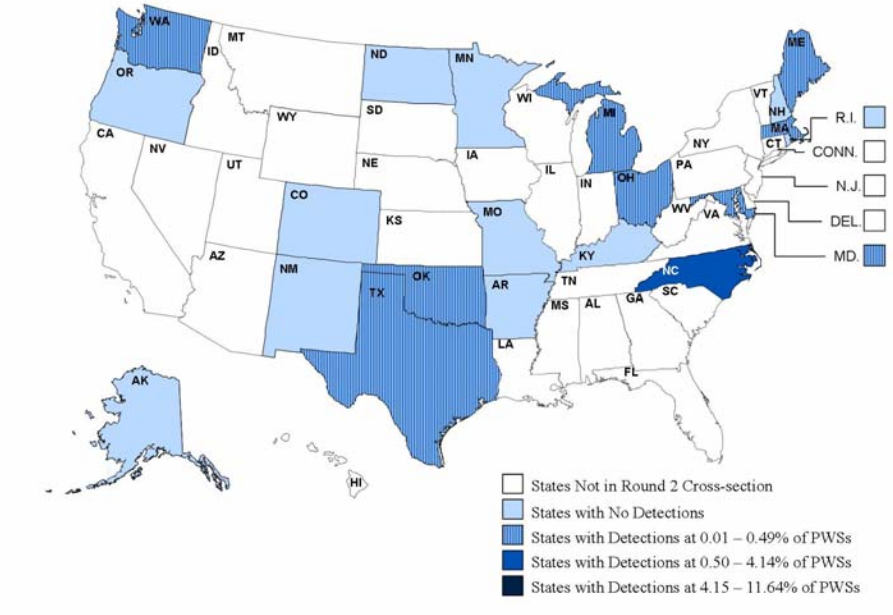
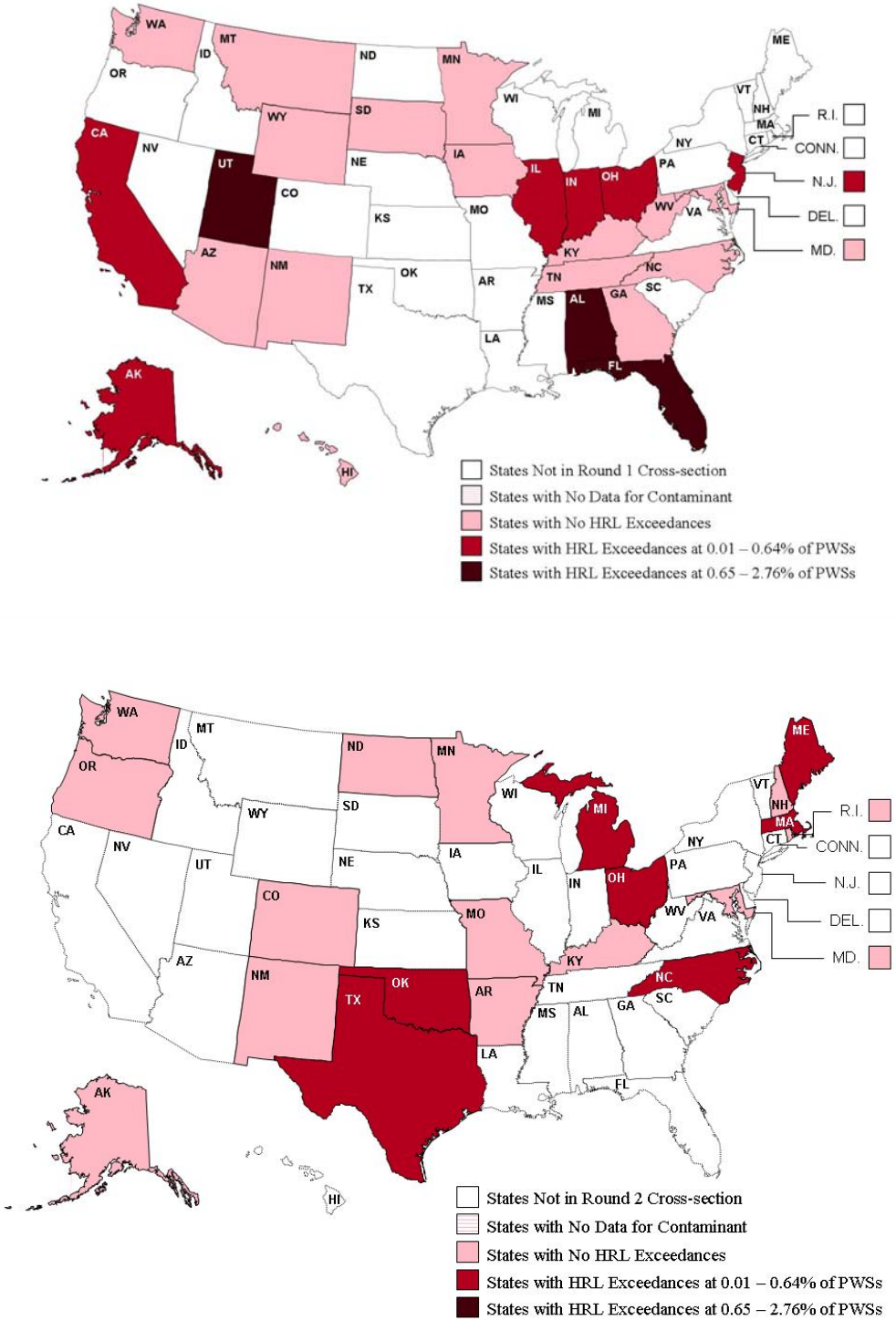


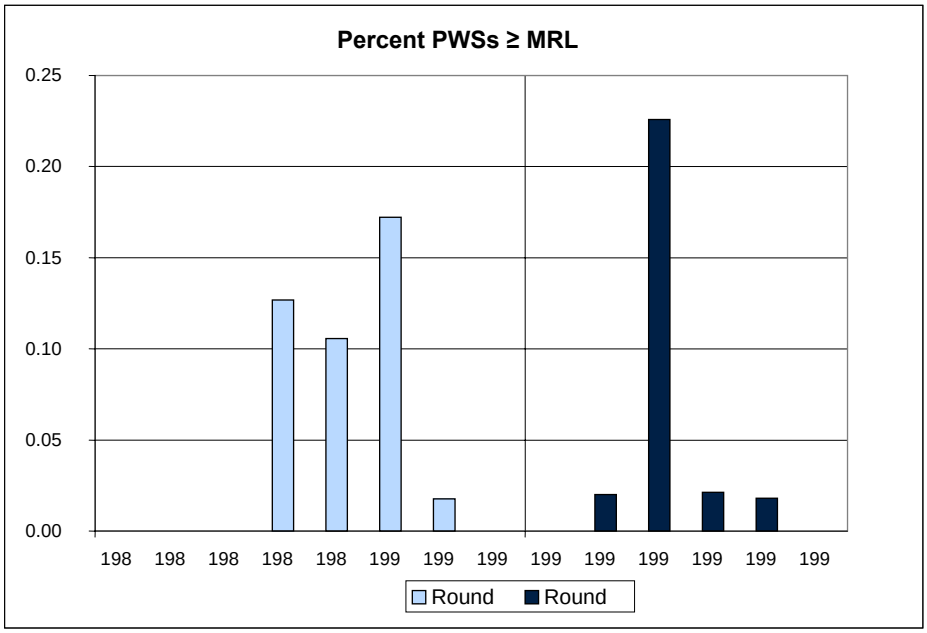
Exhibit 11-9: Geographic Distribution of 1,1,2,2-Tetrachloroethane HRL Exceedance Frequencies in Cross-Section States (Above: UCM Round 1; Below: UCM Round 2)

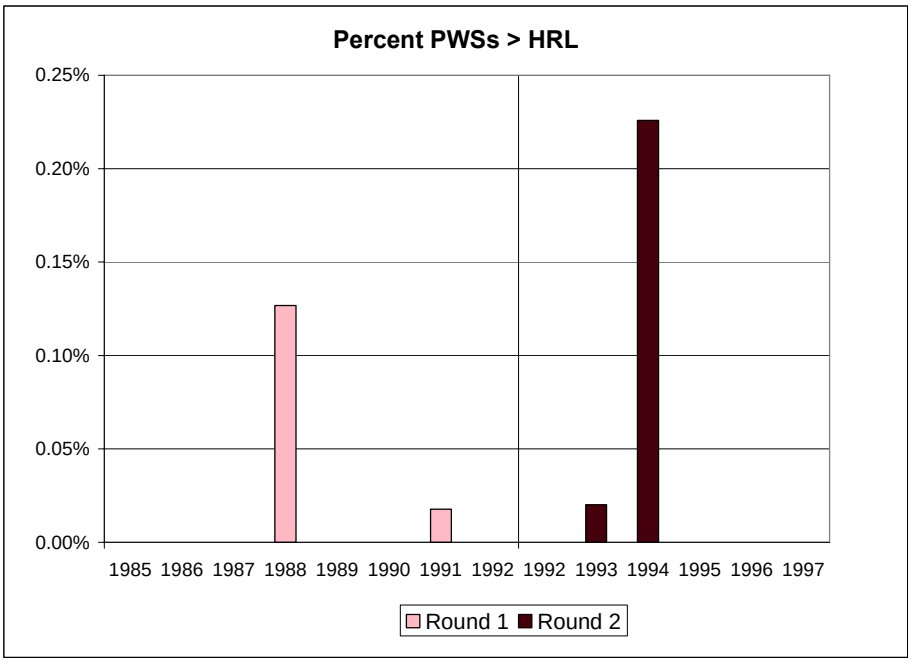


Eight States (AK, KY, MD, MN, NM, NC, OH, and WA) contributed 1,1,2,2-tetrachloroethane data to both the Round 1 and Round 2 cross-sections. While these States are

not necessarily nationally representative, they enable a preliminary assessment of temporal trends in 1,1,2,2-tetrachloroethane occurrence. Exhibits 11-10 and 11-11 suggest that detections in those States were most common in 1988-1990, and again in 1994. HRL exceedances were also most common in 1988 and 1994. Only three of the eight States had detections in both Rounds, and only one State (Ohio) had HRL exceedances in both Rounds.

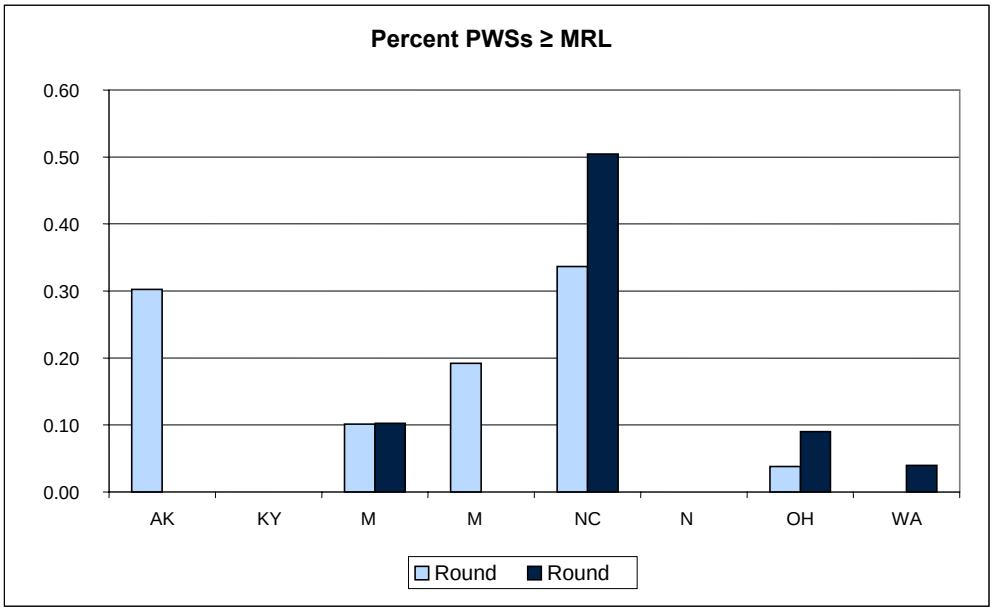
Exhibit 11-10: Annual Frequency of 1,1,2,2-Tetrachloroethane Detections (above) and HRL Exceedances (below), 1985 - 1997, in Select Cross-Section States

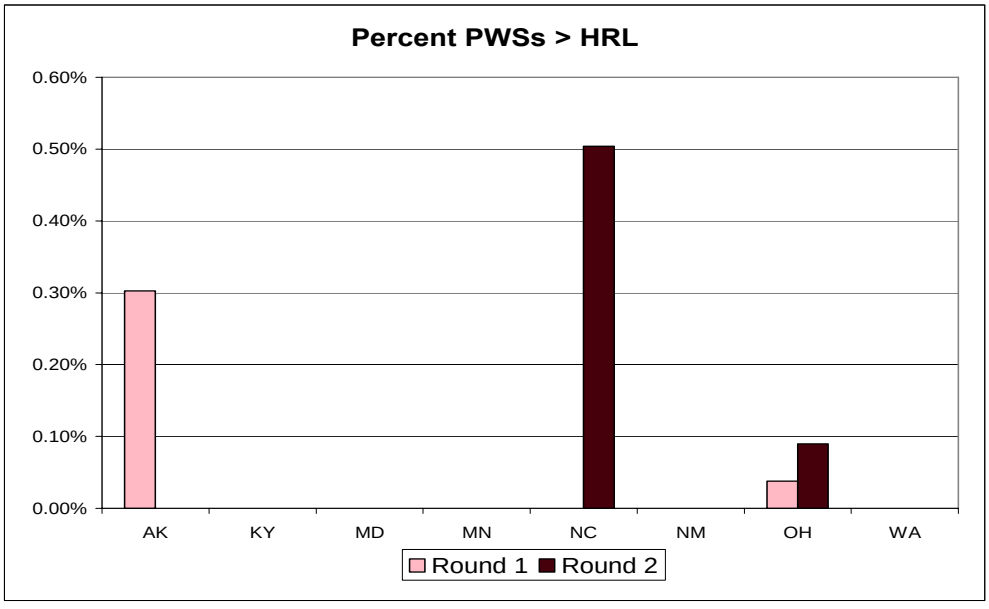




Notes: Data are from AK, KY, MD, MN, NC, NM, OH, and WA. (These eight States are the only States in both the Round 1 and Round 2 cross-sections.) Both Round 1 and Round 2 have data for 1992; 1992 results from each Round are presented separately. The HRL for 1,1,2,2-tetrachloroethane is 0.4 µg/L.

Exhibit 11-11: Distribution of 1,1,2,2-Tetrachloroethane Detections (above) and HRL Exceedances (below) Among Select Cross-Section States





Notes: These eight States are the only States in both the Round 1 cross-section and the Round 2 cross-section. The HRL for 1,1,2,2-tetrachloroethane is 0.4 µg/L.

11.4 Technology Assessment

11.4.1 Analytical Methods

Two analytical methods are available for detecting 1,1,2,2-tetrachloroethane in drinking water. EPA Methods 502.2 and 524.2 rely on purge and trap gas chromatography (GC) followed by either electrolytic conductivity detection (ELCD) or mass spectrometry (MS). A description of these methods can be found in EPA's *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III,* available from the Drinking Water Public Docket or the National Technical Information Service (USEPA, 1995a). Historically, Methods 502.1 and 524.1 were also used to collect occurrence data for 1,1,2,2-tetrachloroethane. These methods are based on similar technology to Methods 502.2 and 524.2, but are now considered obsolete. Their approval for use for compliance monitoring of VOCs was withdrawn as of July 1, 1996.

The MDL and the average recovery for each analytical method that can be used for the analysis of 1,1,2,2-tetrachloroethane are included in the method descriptions below.[1]

EPA Method 502.2

EPA Method 502.2 (Revision 2.1), entitled "*Volatile Organic Compounds in Water by Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series*," determines the presence of VOCs in water samples using GC with ELCD or photoionization (PID). However, only ELCD can be used for 1,1,2,2-tetrachloroethane analysis, as this compound does not respond to PIDs.

The method detection limit (MDL) for 1,1,2,2-tetrachloroethane using this method is reported to range from 0.01 to 0.02 µg/L, and the average recovery is reported to range from 99 to 100 percent, depending on the method option used (USEPA, 1995b).

EPA Method 524.2

EPA Method 524.2 (Revision 4.1), "*Measurement of Purgeable Organic Compounds in Water by Capillary Column Gas Chromatography/Mass Spectrometry*," is used to detect VOCs, including 1,1,2,2-tetrachloroethane, in finished drinking water, raw source water, or drinking water in any treatment stage.

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[1] The Method Detection Limit (MDL) is a statistical estimate of the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analyte concentration is greater than zero, *i.e.*, greater than the background signal. The calculation of the MDL is based upon the precision of a series of replicate measurements of the analyte at low concentrations. The MDL incorporates estimates of the accuracy of the determination. The MDL is not a concentration that can typically be measured by the method on a routine basis. Detection limits may vary between analysts and laboratories under various laboratory conditions.

The average recovery is the fraction or percent concentration of a target analyte determined relative to the true or expected concentration from a sample containing a known amount of the target analyte. (This can result in apparent recovery values greater than 100 percent.)

VOCs such as 1,1,2,2-tetrachloroethane are extracted by bubbling an inert gas through the aqueous sample. Purged sample components are trapped in a tube containing suitable sorbent materials. When purging is complete, the sorbent tube is heated and backflushed with helium to thermally desorb trapped sample components onto a capillary GC column. The column is temperature-programmed to separate the method analytes, which are then detected with a mass spectrometer. Analytes are identified and quantitated by comparison to standard materials (USEPA, 1995c).

The MDL for 1,1,2,2-tetrachloroethane using this method is reported to range from 0.04 to 0.2 µg/L, and the average recovery is reported to range from 91 to 100 percent, depending on the method option used (USEPA, 1995c).

11.4.2 Treatment Technologies

Treatment technology status does not influence the determination of whether or not a contaminant should be regulated. However, treatment technologies must be readily available before a contaminant can be regulated with a national primary drinking water regulation (NPDWR). Potential treatment technologies for removing 1,1,2,2-tetrachloroethane include air stripping and activated carbon.

Air stripping involves the continuous contact of air with the water being treated, allowing dissolved volatile contaminants to transfer from the source water to the air. Systems often consist of a large column (or tower) filled with molded plastic or ceramic packing material. As the water flows along the column, air is forced counter-current through the water. The packing material increases the area of air-liquid interface, enhancing mass transfer. After contact, the air is vented to an additional treatment device that safely contains or destroys the contaminant.

The Henry's Law constant is commonly used to indicate the tendency of a contaminant to partition from water to air. A larger Henry's constant indicates a greater equilibrium concentration of the contaminant in the air. A compound is generally considered amenable to air stripping if it has a Henry's constant above that of dibromochloropropane (0.003 mol/mol) or ethylene dibromide (0.013 mol/mol) (Speth *et al.*, 2001). Speth *et al.* (2001) compiled Henry's Law constants, both calculated by the authors and reported in the literature, for Contaminant Candidate List (CCL) compounds. These authors report Henry's Law constants of 0.012 mol/mol and 0.016 mol/mol for 1,1,2,2-tetrachloroethane, suggesting that air stripping might be a viable treatment option (Speth *et al.*, 2001).

Granular activated carbon (GAC) treatment removes contaminants via the physical and chemical process of sorption: the contaminants attach to the carbon surface as water passes through the carbon bed. Activated carbon has a large sorption capacity for many water impurities, including synthetic organic chemicals, taste- and odor-causing compounds, and some species of mercury.

Adsorption capacity is typically represented by the Freundlich isotherm constant, with higher Freundlich (K) values indicating greater sorption potential. Activated carbon is considered to be cost-effective for removing a particular contaminant if the Freundlich (K) value of the contaminant is above 200 µg/g $(L/\mu g)^{1/n}$ (Speth *et al.*, 2001). Speth and Adams (1993 as

cited in Speth *et al.*, 2001) report that the Freundlich (K) value for 1,1,2,2-tetrachloroethane is 823 μg/g $(L/\mu g)^{1/n}$, which indicates that GAC might be a viable treatment option.

11.5 Regulatory Determination

The Agency has made a preliminary determination not to regulate 1,1,2,2-tetrachloroethane with an NPDWR. Because 1,1,2,2-tetrachloroethane appears to occur infrequently at health levels of concern in PWSs, the Agency believes that an NPDWR does not present a meaningful opportunity for health risk reduction. While 1,1,2,2-tetrachloroethane was detected in both the UCM Round 1 and the UCM Round 2 surveys, the percentage of detections had decreased by the time the UCM Round 2 survey was performed in the mid-1990's. In addition, the USGS did not detect 1,1,2,2-tetrachloroethane in two subsequent monitoring surveys of source waters that supply community water systems using a reporting limit that is less than the 1,1,2,2-tetrachloroethane HRL. The Agency believes that this decrease in detections occurred because commercial production of 1,1,2,2-tetrachloroethane ceased in the mid-1980's. Hence, the Agency does not expect 1,1,2,2-tetrachloroethane to occur in many public water systems today.

EPA recognizes that 1,1,2,2-tetrachloroethane is listed as a likely human carcinogen. For this reason, the Agency encourages those States with public water systems that may have 1,1,2,2-tetrachloroethane above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory document for 1,1,2,2-tetrachloroethane to provide more recent health information. The updated Health Advisory will provide information to any States with public water systems that may have 1,1,2,2-tetrachloroethane at levels above the HRL.

The Agency's preliminary regulatory determination for this contaminant is presented formally in the *Federal Register*.

11.6 References

Agency for Toxic Substances and Disease Registry (ATSDR). 1996. *Toxicological Profile for 1,1,2,2-Tetrachloroethane*. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services. Available on the Internet at: http://www.atsdr.cdc.gov/toxprofiles/tp93.html.

Archer, W.L. 1979. *Kirk-Othmer Encyclopedia of Chemical Technology*. 3rd ed, Volume 5. Grayson, H. and D. Eckroth, eds. (As cited in ATSDR, 1996).

Assessment Tools for the Evaluation of Risk (ASTER). 1995. ASTER Ecotoxicity Profile. Duluth, MN: Environmental Research Laboratory, USEPA. October 11, 1995. (As cited in ATSDR, 1996).

Atkinson, R. 1987. A structure-activity relationship for the estimation of rate constants for gas-phase reaction of OH radicals with organic compounds. *International Journal of Chemical Kinetics*. 19:799-828. (As cited in ATSDR, 1996).

Baldys, S, III, T.H. Raines, B.L. Mansfield, and J.T. Sandlin. 1998. *Urban stormwater quality, event mean concentrations, and estimates of stormwater pollutant loads, Dallas-Fort Worth area, Texas, 1992–93.* U.S. Geological Survey Water-Resources Investigation Report 98-4158, 51 p.

Bouwer, E.J. and P.L McCarty. 1983. Transformations of 1-Carbon and 2-Carbon Halogenated Aliphatic Organic-Compounds Under Methanogenic Conditions. *Applied and Environmental Microbiology*. 45:1286-1294. (As cited in Howard, 1990).

Cole, R.H., R.E. Frederick, R.P. Healy, and R.G. Rolan. 1984. Preliminary findings of the priority pollutant monitoring project of the Nationwide Urban Runoff Program. *Journal of the Water Pollution Control Federation*. 56(7):898-908.

Cooper, W.J., M. Mehran, D.J. Riusech, and J.A. Joens. 1987. Abiotic transformations of halogenated organics. 1. Elimination reaction of 1,1,2,2-tetrachloroethane and formation of 1,1,2-trichloroethene. *Environmental Science and Technology.* 21:1112-1114. (As cited in ATSDR, 1996).

Delzer, G.C. and T. Ivahnenko. 2003. *Occurrence and Temporal Variability of Methyl tert-Butyl Ether (MTBE) and Other Volatile Organic Compounds in Select Sources of Drinking Water: Results of the Focused Survey.* U.S. Geological Survey Water-Resources Investigations Report 02-4084, 65 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4084.pdf.

Gerhartz, W. (exec ed.). 1985. *Ullmann's Encyclopedia of Industrial Chemistry*. 5th ed. Vol A1. Deerfield Beach, FL: VCH Publishers. (As cited in HSDB, 2004).

Grady, S.J. 2003. *A National Survey of Methyl tert-Butyl Ether and Other Volatile Organic Compounds in Drinking-Water Sources: Results of the Random Survey.* U.S. Geological Survey Water-Resources Investigations Report 02-4079, 85 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4079.pdf.

Haag, W.R. and T. Mill. 1988. Effect of a subsurface sediment on hydrolysis of haloalkanes and epoxides. *Environmental Science and Technology*. 22:658-663. (As cited in ATSDR, 1996).

Hallen, R.T., J.W. Pyne Jr., and P.M. Molton. 1986. Transformation of chlorinated ethenes and ethanes by anaerobic microorganisms. In: *192nd National Meeting ACS Division Environmental Chemistry*. pp. 344-346. (As cited in ATSDR, 1996).

Hansch, C., A. Leo, and D. Hoekman. 1995. *Exploring QSAR - Hydrophobic, Electronic, and Steric Constants.* Washington, DC: American Chemical Society. (As cited in HSDB, 2004).

Hawley, G.G. 1981. *Condensed Chemical Dictionary*. 10th ed. New York, NY: Van Nostrand Reinhold Co. (As cited in ATSDR, 1996).

Hazardous Substances Data Bank (HSDB). 2004. Search for 1,1,2,2-tetrachloroethane. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed October 25, 2004.

Horvath, A.L. 1982. *Halogenated Hydrocarbons, Solubility-Miscibility with Water.* NY: Marcel Dekker, Inc. (As cited in HSDB, 2004).

Howard, P.H. 1990. *Handbook of Environmental Fate and Exposure Data for Organic Chemicals. Volume II Solvents*. Chelsea, MI: Lewis Publishers, Inc.

International Agency for Research on Cancer (IARC). 1979. *1,1,2,2-Tetrachloroethane*. IACR Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some halogenated hydrocarbons. Vol. 20. pp. 477-489. (As cited in ATSDR, 1996).

Ivahnenko, T., S.J. Grady, and G.C. Delzer. 2001. *Design of a National Survey of Methyl tert-Butyl Ether and Other Volatile Organic Compounds in Drinking-Water Sources.* U.S. Geological Survey Open-File Report 01-271, 42 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr01_271.pdf.

Jeney, E., F. Bartha, L. Kondor, and S. Szendrei. 1957. Prevention of industrial tetrachloroethane intoxication--Part III. *Egeszsegtudomany*. 1:155-164. (As cited in USEPA, 1989).

Konietzko, H. 1984. Chlorinated ethanes: Sources, distribution, environmental impact, and health effects. *Hazard Assessment of Chemicals Current Developments*. 3:401-448. (As cited in ATSDR, 1996).

Lehman, K.B. and L. Schmidt-Kehl. 1936. [Study of the 13 most important chlorohydrocarbons from the standpoint of industrial hygienics]. *Archiv Fuer Hygiene und Bakteriologie*. 116:132-268. (As cited in ATSDR, 1996 and USEPA, 1989).

Lide, D.R. (ed.). 1995. *CRC Handbook of Chemistry and Physics. 75th ed.* Boca Raton, Fl: CRC Press Inc., 1994-1995. (As cited in HSDB, 2004).

Lobo-Mendonca, R. 1963. Tetrachloroethane - A survey. *British Journal of Industrial Medicine*. 20:51-56. (As cited in ATSDR, 1996 and USEPA, 1989).

Lopes, T.J., K.D. Fossum, J.V. Phillips, and J.E. Monical. 1995. *Statistical summary of selected physical, chemical, and microbial characteristics, and estimates of constituent loads in urban stormwater, Maricopa County, Arizona.* U.S. Geological Survey Water-Resources Investigations Report 94-4240.

Lopes, T.J. and S.G. Dionne. 1998. *A Review of Semivolatile and Volatile Organic Compounds in Highway Runoff and Urban Stormwater.* U.S. Geological Survey Open-File Report 98-409, 67 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr98-409.pdf.

Minot, G.R. and L.W. Smith. 1921. The blood in tetrachloroethane poisoning. *Archives of Internal Medicine*. 28:687-702. (As cited in ATSDR, 1996).

National Cancer Institute (NCI). 1978. *Bioassay of 1,1,2,2-Tetrachloroethane for Possible Carcinogenicity.* NCI-CG-TR-27. (As cited in ATSDR, 1996).

National Toxicity Program (NTP). 1991a. *Range Finding Studies: Developmental Toxicity — 1,1,2,2-Tetrachloroethane When Administered via Feed in CD Sprague-Dawley Rats.* NTP-91-RF/DT-017.

NTP. 1991b. *Range Finding Studies: Developmental Toxicity — 1,1,2,2-Tetrachloroethane (Repeat) When Administered via Feed in Swiss CD-1 Mice.* NTP-91-RF/DT-020.

NTP. 2004. *NTP technical report on the toxicity studies of 1,1,2,2-tetrachloroethane administered in microcapsules in feed to F-344/N rats and B6C3F1 mice.* NIH Publication No. 04-4414.

Schmidt P., S. Binnevies, R. Gohlke, and R. Roth. 1972. Subacute action of low concentration of chlorinated ethanes on rats with and without additional ethanol treatment. I. Biochemical and toxicometrical aspects, especially results in subacute and chronic toxicity studies with 1,1,2,2-tetrachloroethane. *Internationales Archiv Fur Arbeitsmedizin.* 30:283-298.

Singh, H.B., L.J. Salas, A.J. Smith, and H. Shigeishi. 1981. Measurements of some potentially hazardous organic chemicals in urban environments. *Atmospheric Environment*. 15:601-612. (As cited in HSDB, 2004).

Speth, T.F. and J.Q. Adams. 1993. GAC and Air Stripping Design Support for the Safe Drinking Water Act. *Strategies and Technologies for Meeting SDWA Requirements*. Clark, R. and S. Summers, Eds., Lewis Publishers, Ann Arbor, MI, pp. 47-89. (As cited in Speth *et al*., 2001).

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

Squillace, P.J., M.J. Moran, W.W. Lapham, C.V. Price, R.M. Clawges, and J.S. Zogorski. 1999. Volatile organic compounds in untreated ambient groundwater of the United States, 1985-1995. *Environmental Science and Technology*. 33(23):4176-4187. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/journal/EST.voc.squillace.pdf.

United States Environmental Protection Agency (USEPA). 1989. *1,1,2,2-Tetrachloroethane Health Advisory*. May, 1989.

USEPA. 1995a. *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III*. EPA Report 600-R-95-131. August, 1995.

USEPA. 1995b. Volatile Organic Compounds in Water by Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series. Revision 2.1. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III*. EPA Report 600-R-95-131. August, 1995.

USEPA. 1995c. Measurement of Purgeable Organic Compounds in Water by Capillary Column Gas Chromatography/Mass Spectrometry. Revision 4.1. In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement III*. EPA Report 600-R-95-131.

USEPA. 2006a. TRI Explorer: Trends. Search for 1,1,2,2-tetrachloroethane. Available on the Internet at: http://www.epa.gov/triexplorer/trends.htm. Accessed February 8, 2006. Last modified June 8, 2005.

USEPA. 2006b. *The Analysis of Occurrence Data from the Unregulated Contaminant Monitoring (UCM) Program and National Inorganics and Radionuclides Survey (NIRS) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-009. Draft. December 2006.

von Guerard, P. and W.B. Weiss. 1995. *Water quality of storm runoff and comparison of procedures for estimating storm-runoff loads, volume, event-mean concentrations, and the mean load for a storm for selected properties and constituents for Colorado Springs, southeastern Colorado, 1992*. U.S. Geological Survey Water-Resources Investigations Report 94-4194.

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

Part III: What About the Remaining CCL 2 Contaminants?

As stated in Chapter 1, EPA is only making regulatory determinations on CCL 2 contaminants that have sufficient information to support a regulatory determination at this time. EPA is not able to make a preliminary determination for perchlorate at this time, because in order to evaluate perchlorate against the three statutory criteria, the Agency believes additional information may be needed to more fully characterize perchlorate exposure and determine whether regulating perchlorate in drinking water presents a meaningful opportunity for health risk reduction. For the 30 remaining chemicals and the 9 microbial pathogens, the Agency lacks adequate information in the areas of health effects or occurrence or both.

The Agency continues to conduct research and/or to collect information on the remaining high-priority contaminants to fill identified data gaps. Stakeholders may be concerned that regulatory determinations for such contaminants should not necessarily wait until the end of the next regulatory determination cycle. In this regard, it is important to recognize that the Agency is not precluded from conducting research, monitoring, developing guidance or health advisories, and/or making a determination prior to the end of the next cycle. In addition, the Agency is not precluded from regulating a contaminant at any time when it is necessary to address an urgent threat to public health, including any contaminant not listed on the CCL.

Of the remaining CCL 2 contaminants, the Agency recognizes that the public may have a particular interest in perchlorate, metolachlor, methyl tertiary butyl ether (MTBE), and the microbial contaminants. Therefore, this report includes some additional information for these contaminants in the following sections.

Chapter 12: Perchlorate

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Contents

Exhibits

Abbreviations

AWS	American Water System
AWWARF	American Water Works Association Research Foundation
CA DHS	California Department of Health Services
CAS	Chemical Abstracts Service
CCL	Contaminant Candidate List
CDC	Centers for Disease Control
DW	Dry Weight
DWEL	Drinking Water Equivalent Level
FDA	Food and Drug Administration
FW	Fresh Weight
HRL	Health Reference Level
IC	Ion Chromatography
IC/MS	Ion Chromatography Mass Spectrometry
IC-MS/MS	Ion Chromatography Tandem Mass Spectrometry
IC-ESI-MS/MS	Ion Chromatography Electrospray Ionization Tandem Mass Spectrometry
IOC	Inorganic Compound
IRIS	Integrated Risk Information System
LC-MS/MS	Liquid Chromatography Tandem Mass Spectrometry
MA DEP	Massachusetts Department of Environmental Protection
MRL	Minimum Reporting Level or Minimum Reporting Limit
MS/MS	Tandem Mass Spectrometry
NAS	National Academies of Science
NCEH	National Center for Environmental Health
ND	Not Detected
NHANES	National Health and Nutrition Examination Survey
NIS	Sodium (Na^+) Iodide (I^-) Symporter
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
NQ	Not Quantifiable
NRC	National Research Council
PWS	Public Water System
RDA	Recommended Daily Allowance
RfD	Reference Dose
RSC	Relative Source Contribution
TDS	Total Diet Study
TSH	Thyroid-Stimulating Hormone
UCMR 1	First Unregulated Contaminant Monitoring Regulation
USDA	United States Department of Agriculture
USDA-ARS	U.S. Department of Agriculture – Agricultural Research Service
WHO	World Health Organization

12 Perchlorate

12.1 Definition

Perchlorate is an inorganic contaminant (IOC) containing one chlorine atom bound to four oxygen atoms in a tetrahedral configuration. As such, perchlorate (ClO_4^-) is a group of anions that forms salts with most cations. Commonly used perchlorate salts include ammonium perchlorate and potassium perchlorate. Perchlorate is also used as sodium perchlorate, aluminum perchlorate, hydrazine perchlorate, hydrogen perchlorate, hydroxylammonium perchlorate, lithium perchlorate, magnesium perchlorate, nitronium perchlorate, and as perchloric acid. As an anion, there is no single Chemical Abstracts Service (CAS) registry for perchlorate, as each salt has its own properties. Registry numbers for the most common forms of perchlorate are presented in Exhibit 12-1.

12.1.1 Properties and Sources

Perchlorate (ClO_4^-) is an anion commonly associated with the solid salts of ammonium, magnesium, potassium, and sodium perchlorate. Although commonly known as a man-made chemical, perchlorate also may be derived from natural processes.

Chile possesses caliche ores rich in sodium nitrate ($NaNO_3$), which are also a natural source of perchlorate (Schilt, 1979 and Ericksen, 1983, as cited in USEPA, 2001). These Chilean nitrate salts (saltpeter) have been mined and refined to produce commercial fertilizers, which before 2001 accounted for about 0.14 percent of U.S. fertilizer application (USEPA, 2001). Perchlorate has also been found in other geologic materials. Orris *et al.* (2003) measured perchlorate at levels exceeding 1,000 parts per million (ppm or mg/kg) in several samples of natural minerals, including potash ore from New Mexico and Saskatchewan (Canada), playa crust from Bolivia, and hanksite from California.

Texas Tech University Water Resources Center conducted a large-scale sampling program to determine the source and distribution of perchlorate in northwest Texas groundwater (Jackson *et al.*, 2004; Rajagopalan *et al.*, 2006). Perchlorate was detected at concentrations greater than 0.5 µg/L in 46 percent of public wells and 47 percent of private wells. Jackson *et al.* (2004) hypothesized that atmospheric production and/or surface oxidative weathering is the source of the perchlorate. In related research, Dasgupta *et al.* (2005) detected perchlorate in many rain and snow samples and demonstrated that perchlorate is formed by a variety of simulated atmospheric processes suggesting that natural, atmospherically-derived perchlorate exists in the environment. Barron *et al.* (2006) developed a method for the rapid determination of perchlorate in rainwater samples, with a detection limit between 70 and 80 ng/L. Of the ten rainwater samples collected in Ireland in 2005, perchlorate was detected in 4 samples at concentrations between 0.075 and 0.113 µg/L, and in 1 other sample at 2.8 µg/L. Kang *et al.* (2006) conducted seven-day experiments to determine if it was possible to produce perchlorate by exposing various chlorine intermediates to UV radiation in the form of high intensity UV lamps and/or ambient solar radiation. Perchlorate formation was demonstrated in aqueous salt solutions with initial concentrations of hypochlorite, chlorite, or chlorate between 100 and 10,000 mg/L.

After a limited investigation, the Massachusetts Department of Environmental Quality (MA DEP, 2005) found that perchlorate may be present in sodium hypochlorite solutions used in water and wastewater treatment plants, and that the level of occurrence depends upon storage conditions and the initial purity of the stock solution (MA DEP, 2005). According to MA DEP (2005), the Town of Tewksbury conducted a small study to evaluate the impact of storage conditions (temperature and light) on a new shipment of sodium hypochlorite stock solution. Tewksbury found that the perchlorate concentration in the new stock solution increased from 0.2 µg/L to levels ranging from 995 to 6,750 µg/L depending on the storage conditions. Accounting for the large dilution factor (e.g., 20,000 to 1 ratio) used in chlorination processes at drinking water treatment plants, MA DEP (2005) concluded that "absent additional efforts to minimize breakdown of hypochlorite solutions, it would appear that low levels of the perchlorate ion (0.2 to 0.4 µg/L) detected in a drinking water supply disinfected with sodium hypochlorite solutions could be attributable to the chlorination process."

It is not clear at this time what proportion of perchlorate found in public water supplies or entering the food chain comes from these various anthropogenic and natural sources. The significance of different sources probably varies regionally. A study by Dasgupta et al. (2006) analyzes the three principal sources of perchlorate and their relative contributions to the food chain. These are its use as an oxidizer including rocket propellants, Chilean nitrate used principally as fertilizer, and that produced by natural atmospheric processes.

Some physical and chemical properties of perchlorate and common perchlorate salts are listed in Exhibit 12-1.

Exhibit 12-1: Physical and Chemical Properties

Perchlorate and Its Common Salts					
	Perchlorate	**Ammonium perchlorate**	**Potassium perchlorate**	**Magnesium perchlorate**	**Sodium perchlorate**
CAS number	14797-73-0	7790-98-9	7778-74-7	10034-81-8	7601-89-0
Molecular Formula	ClO_4^-	NH_4ClO_4	$KClO_4$	$Mg(ClO_4)_2$	$NaClO_4$
Physical and Chemical Properties					
Boiling Point	-----	-----	400 °C [1]	-----	-----
Melting Point	-----	439 °C [2]	525 °C [4]	250 °C [4]	480 °C [4]
Molecular Weight	99.45 g/mol [1]	117.49 g/mol [2]	138.55 g/mol [1]	223.20 g/mol [4]	122.4 g/mol [1]
Water Solubility	-----	200 g/L @ 25°C [3]	15 g/L @ 25°C [3]	99 g/1000g @ 25°C [5]	209 g/100 g @ 25 °C [6]

[1] *Budavari, 1996 (as cited in HSDB, 2004)*

[2] *HSDB, 2004*

[3] *Ashford, 1994 (as cited in HSDB, 2004)*

[4] *Lide, 2000 (as cited in HSDB, 2004)*

[5] *Weast, 1979 (as cited in HSDB, 2004)*

[6] *Gerhartz, 1985 (as cited in HSDB, 2004)*

12.1.2 Environmental Fate and Behavior

Perchlorate salts are highly soluble in water, and because perchlorate sorbs poorly to mineral surfaces and organic material, perchlorate can be mobile in surface and subsurface aqueous environments (USEPA, 2002).

12.2 Health Effects

Perchlorate can interfere with the normal functioning of the thyroid gland by competitively inhibiting the transport of iodide into the thyroid. Iodide is an important component of two thyroid hormones, T4 and T3, and the transfer of iodide from the blood into the thyroid is an essential step in the synthesis of these two hormones. Iodide transport into the thyroid is mediated by a protein molecule known as the sodium (Na+) – iodide (I-) symporter (NIS). NIS molecules bind iodide with very high affinity, but they also bind other ions that have a similar shape and electric charge, such as perchlorate. The binding of these other ions to the NIS inhibits iodide transport into the thyroid, which can result in intrathyroidal iodide deficiency and consequently decreased synthesis of T4 and T3. There is compensation for iodide deficiency, however, such that the body maintains the serum concentrations of thyroid hormones within narrow limits through feedback control mechanisms. This feedback includes increased secretion of thyroid stimulating hormone (TSH) from the pituitary gland, which has among its effects the increased production of T4 and T3 (USEPA, 2005). Sustained changes in thyroid

hormone and TSH secretion can result in thyroid hypertrophy and hyperplasia (abnormal growth or enlargement of the thyroid) (USEPA, 2005).

In January 2005, the National Research Council (NRC) of the National Academies of Science (NAS) published "Health Implications of Perchlorate Ingestion," a review of the current state of the science regarding potential adverse health effects of perchlorate exposure and mode-of-action for perchlorate toxicity (NRC, 2005). Based on recommendations of the NRC, EPA chose data from the Greer *et al.* (2002) human clinical study as the basis for deriving a reference dose (RfD) for perchlorate (USEPA, 2005). Greer *et al.* (2002) report the results of a well-controlled study that measured thyroid iodide uptake, hormone levels, and urinary iodide excretion in a group of 24 healthy adults administered perchlorate doses orally over a period of 14 days. Dose levels ranged from 0.007 to 0.5 mg/kg/day in the different experimental groups. No significant differences were seen in measured serum thyroid hormone levels (T3, T4, total and free) in any dose group. The statistical no-observed-effect level (NOEL) for perchlorate-induced inhibition of thyroid iodide uptake was 0.007 mg/kg/day. Although the NRC committee concluded that hypothyroidism is the first adverse effect in the continuum of effects of perchlorate exposure, NRC recommended that "the most health-protective and scientifically valid approach" was to base the perchlorate RfD on the inhibition of iodide uptake by the thyroid (NRC, 2005). NRC concluded that iodide uptake inhibition, although not adverse, is the key biochemical event in the continuum of possible effects of perchlorate exposure and would precede any adverse health effects of perchlorate exposure. The lowest dose (0.007 mg/kg/day) administered in the Greer *et al.* (2002) study was considered a NOEL (rather than a "no-observed-adverse-effect level" or NOAEL) because iodide uptake inhibition is not an adverse effect but a biochemical change (USEPA, 2005). A summary of the data considered and the NRC deliberations can be found in the NRC report (2005) and the EPA Integrated Risk Information System (IRIS) summary (USEPA, 2005).

The NRC recommended that EPA apply an intraspecies uncertainty factor of 10 to the NOEL to account for differences in sensitivity between the healthy adults in the Greer *et al.* (2002) study and the most sensitive population, fetuses of pregnant women who might have hypothyroidism or iodide deficiency. Because the fetus depends on an adequate supply of maternal thyroid hormone for its central nervous system development during the first trimester of pregnancy, iodide uptake inhibition from low-level perchlorate exposure has been identified as a concern in connection with increasing the risk of neurodevelopmental impairment in fetuses of high-risk mothers (NRC, 2005). The NRC (2005) viewed the uncertainty factor of 10 as conservative and health protective given that the point of departure is based on a non-adverse effect (iodide uptake inhibition) that precedes the adverse effect in a continuum of possible effects of perchlorate exposure. NRC concluded that no uncertainty factor was needed for the use of a less-than chronic study, for deficiencies in the database, or for interspecies variability. To protect the most sensitive human population from chronic perchlorate exposure, EPA derived an RfD of 0.0007 mg/kg/day with a ten-fold total uncertainty factor from the NOEL of 0.007 mg/kg/day (USEPA, 2005).

Blount *et al.* (2006b) recently published a study examining the relationship between urinary levels of perchlorate and serum levels of TSH and total T4 in 2,299 men and women (ages 12 years and older), who participated in the Centers for Disease Control's (CDC's) 2001-

2002 National Health and Nutrition Examination Survey (NHANES)[1]. Blount *et al.* (2006b) evaluated perchlorate along with covariates known or likely to be associated with T4 or TSH levels to assess the relationship between perchlorate and these hormones, and the influence of other factors on this relationship. These covariates included sex, age, race/ethnicity, body mass index, serum albumin, serum cotinine (a marker of tobacco smoke exposure), estimated total caloric intake, pregnancy status, post-menopausal status, premenarche status, serum C-reactive protein, hours fasting before sample collection, urinary thiocyanate, urinary nitrate, and use of selected medications. The study found that perchlorate was a significant predictor of thyroid hormones in women, but not men.

After finding evidence of gender differences, the researchers focused on further analyzing the NHANES data for the 1,111 women participants. They divided these 1,111 women into two categories, higher-iodide and lower-iodide, using a cut point of 100 µg/L of urinary iodide based on the World Health Organization (WHO) definition of sufficient iodide intake[2]. Hypothyroid women were excluded from the analysis. According to the study authors, about 36 percent of women living in the United States have urinary iodide levels less than 100 µg/L (Caldwell *et al.*, 2005). For women with urinary iodide levels less than 100 µg/L, the study found that urinary perchlorate is associated with a decrease in (a negative predictor for) T4 levels and an increase in (a positive predictor for) TSH levels. For women with urinary iodide levels greater than or equal to 100 µg/L, the researchers found that perchlorate is a significant positive predictor of TSH but not a predictor of T4. The study found that perchlorate was not a significant predictor of T4 or TSH in men. The researchers state that perchlorate could be a surrogate for another unrecognized determinant of thyroid function. Also, the study reports that while large doses of perchlorate are known to decrease thyroid function, this is the first time an association of decreased thyroid function has been observed at these low levels of perchlorate exposure. Of note is that the vast majority of the participants in this group had urinary levels of perchlorate corresponding to estimated dose levels that are below the RfD of 0.0007 mg/kg/day.

The clinical significance of the variations in T4/TSH levels, which were generally within normal limits, has not been determined. The researchers noted several limitations of the study (e.g., assumption that urinary perchlorate correlates with perchlorate levels in the stroma and tissue and preference for measurement of free T4 as opposed to total T4) and recommended that these findings be confirmed in at least one more large study focusing on women with low urine iodide levels. It is also not known whether the association between perchlorate and thyroid hormone levels is causal or mediated by some other correlate of both, although the relationship between urine perchlorate and total TSH and T4 levels persisted after statistical adjustments for some additional covariates known to predict thyroid hormone levels (e.g., total kilocalorie intake, estrogen use, and serum C-reactive protein levels). A planned follow-up study will include additional measures of thyroid health and function (e.g. TPO-antibodies, free T4). As EPA proceeds towards a regulatory determination for perchlorate, the Agency will continue to review any new findings/studies on perchlorate and their relationship to thyroid function as they become available.

1 While CDC researchers measured urinary perchlorate concentration for 2,820 NHANES participants, TSH and total T4 serum levels were only available for 2,299 of these participants.

2 WHO notes that the prevalence of goiter begins to increase in populations with a median iodide intake level below 100 µg/L (WHO, 1994).

12.3 Occurrence and Exposure

12.3.1 Use and Environmental Release

While perchlorate has a wide variety of industrial uses, it is primarily used in the form of ammonium perchlorate as an oxidizer in solid fuels used to power rockets, missiles, and fireworks. Approximately 90 percent of perchlorate is manufactured for this application (Wang *et al.*, 2002). Perchlorate can also be present as an ingredient or as an impurity in road flares, lubricating oils, matches, aluminum refining, rubber manufacturing, paint and enamel manufacturing, leather tanning, paper and pulp processing (as an ingredient in bleaching powder), and as a dye mordant.

Reports produced by USEPA (2002) and the American Water Works Association Research Foundation (AWWARF) (Wang *et al.*, 2002) summarize publicly available information on industrial production, consumption, and disposal of perchlorate.

As noted above, Chilean nitrate salts (saltpeter) have been mined and refined to produce commercial fertilizers. Before 2001, these accounted for about 0.14 percent of U.S. fertilizer application (USEPA, 2001). The USEPA (2001) conducted a broad survey of fertilizers and other raw materials and found that all products surveyed were devoid of perchlorate except for those known to contain or to be derived from mined Chilean saltpeter.

12.3.2 Ambient Water Occurrence

Ambient waters are lakes, rivers, and aquifers that serve as sources for drinking water. Limited national data on the occurrence of perchlorate in ambient surface and ground water have been compiled by EPA Region 9, are posted on the Internet by the Defense Environmental Network & Information Exchange (DENIX, 2004).

12.3.3 Drinking Water Occurrence

Nationally representative data on perchlorate occurrence in drinking water have been collected by large and small public water systems in accordance with EPA's first Unregulated Contaminant Monitoring Regulation (UCMR 1). For details on the UCMR 1, see Chapter 2 and USEPA (2006). Additional monitoring has been performed by other entities.

UCMR 1

EPA included perchlorate as an analyte in the 1999 Unregulated Contaminant Monitoring Regulation (UCMR 1) and collected drinking water occurrence data for perchlorate from 3,858 public water systems (PWSs) between 2001 and 2005. EPA analyzed the available UCMR 1 data on perchlorate at concentrations greater than or equal to 4 µg/L, the minimum reporting level (MRL) for EPA Method 314.0[3]. The Agency found that approximately 4.1 percent (or 160) of 3,858 PWSs that sampled and reported under UCMR 1 had at least 1 analytical detection of perchlorate (in at least 1 entry/sampling point) at levels greater than or equal to 4 µg/L. These

3 EPA Method 314.0 was the analytical method approved and used for UCMR 1 at the time of data collection.

160 systems are located in 26 states and 2 territories. Of these 160 PWSs, 8 are small systems (serving 10,000 or fewer people) and 152 are large systems (serving more than 10,000 people). Approximately 1.9 percent (or 637) of the 34,193 samples collected (by these 3,858 PWSs) had positive detections of perchlorate at levels greater than or equal to 4 µg/L. The maximum reported concentration of perchlorate was 420 µg/L, which was found in a surface water sample from a PWS in Puerto Rico. The average concentration of perchlorate for those samples with positive detections for perchlorate was 9.85 µg/L and the median concentration was 6.40 µg/L.

These 160 PWSs (with at least 1 analytical detection for perchlorate at levels greater than or equal to 4 µg/L) serve approximately 7.5 percent (or 16.8 million) of the 225 million people served by the 3,858 PWSs that sampled and reported results under UCMR 1. The 16.8 million population-served value represents the total number of people served by the 160 PWSs with at least one detect. Not all people served by these systems necessarily have perchlorate in their drinking water. Some of these 160 public water systems have multiple entry points to the distribution system and not all of the entry points sampled had positive detections for perchlorate in the UCMR 1 survey. An alternative approach to the system-level assessment of populations served is to use an assessment at the entry (sampling) point level[4]. EPA does not have population-served values for each entry point at the system level. However, an assessment can be performed by assuming that each entry (or sampling) point at a public water system serves an equal proportion of the total population-served by the system. In other words, for the alternative assessment, the population served by each system is assumed to be equally distributed across all entry (or sampling) points at each system. For example, if a system serves a million people and has 5 entry points, it is assumed that each entry point serves 200,000 people. Using this approach and counting only the population served for the entry points with positive detections (concentrations greater than or equal to 4µg/L), the total population served by these entry points with perchlorate detections is approximately 5 million. Section 12.4 provides the number of systems and population-served estimates for other thresholds of interest.

California Monitoring

The California Department of Health Services (CA DHS) began monitoring for perchlorate in 1997. In 1999, CA DHS began requiring monitoring for perchlorate for drinking water sources that were identified as vulnerable to perchlorate contamination under California's own State monitoring program (i.e., Unregulated Chemicals for which Monitoring is Required). About 60 percent (or 7,100) of all drinking water sources in California (about 12,000) were monitored for perchlorate under the State monitoring program. Between June 2001 and June 2006, CA DHS (2006) reports that 284 (about 4%) of the approximately 7,100 water sources that monitored had at least 2 or more positive detections for perchlorate at concentrations greater than or equal to 4 µg/L (the reporting limit). These 284 sources supply water for 77 drinking water

4 EPA acknowledges that uncertainties exist in the population-served estimates for this alternative assessment since the population for a system is assumed to be equally distributed across the entry points for that system. Because the actual population-served by an entry point is not known, this alternative approach has an equal chance of underestimating or overestimating the actual population-served by entry points with positive detections for perchlorate. In addition, this approach could underestimate the population served that is potentially exposed to perchlorate and overestimate the level of exposure because it can not incorporate the effects of mixing of water between different entry points within the distribution system. This is because the approach cannot account for the dilution that may occur when water that has no detections of perchlorate is mixed within the distribution system with water that has positive detections for perchlorate.

systems (CA DHS, 2006) and represent active and standby sources (and exclude inactive, destroyed, and abandoned sources, and monitoring and agricultural wells) (CA DHS, 2006).

Massachusetts Monitoring

In 2005, the State of Massachusetts's Department of Environment Protection (MA DEP) reported monitoring results for 85 percent (379 of 450) of its community water systems and 86 percent (212 of 250) of its non-transient, non-community water systems. MA DEP found that 9 (1.5%) of the 591 public water systems detected perchlorate at levels greater than or equal to 1 µg/L (the reporting limit used for a modified version of EPA Method 314.0). MA DEP found that the occurrence of perchlorate for these water systems could be traced to the use of blasting agents, military munitions, fireworks, and, to a lesser degree, sodium hypochlorite disinfectant (MA DEP, 2005).

Texas High Plains Monitoring

As noted above, Texas Tech University Water Resources Center conducted a large-scale sampling program to determine the source and distribution of perchlorate in northwest Texas groundwater (Jackson *et al.*, 2004; Rajagopalan *et al.*, 2006). Perchlorate was detected at concentrations greater than 0.5 µg/L in 46 percent of public wells and 47 percent of private wells. Jackson *et al.* (2004) hypothesized that atmospheric production and/or surface oxidative weathering is the source of the perchlorate. Additional results from the same research team are presented in Jackson *et al.* (2005).

Additional Drinking Water Studies

At least two other states have published investigations of perchlorate occurrence in drinking water: Arizona (ADEQ *et al.*, 2004) and New Jersey (NJDWQI, 2005). In addition, AWWARF sponsored a nationwide survey of perchlorate occurrence (Wang *et al.*, 2002). And the American Water System (AWS), which manages dozens of PWSs nationwide, published its own internal survey of perchlorate occurrence in source water (Gullick *et al.*, 2001).

12.3.4 Studies on Perchlorate Occurrence in Foods, Beverages, and Dietary Supplements

The Food and Drug Administration (FDA), the United States Department of Agriculture (USDA), and researchers from academia and industry have studied perchlorate in foods. Some of these studies are described briefly in this section, and also summarized in Exhibit 12-2. EPA has concluded that the sampling results described in this section and Exhibit 12-2 are too limited to characterize food-borne exposure to perchlorate on a national scale. The sampling data are limited in the types of foods sampled, sample sizes, geographic coverage, and/or analytical method adequacy and many were targeted to foods or areas known or likely to have elevated levels of perchlorate. Section 12.5 of this document describes the limitations of the food sampling data and also describes plans for including perchlorate as part of the FDA's Total Diet Study.

Exhibit 12-2: Summary Data on Perchlorate Occurrence in Food Items

Food Item	Data Reference	Units	N	MRL	Range of Detections	Reported Means[s]	Rate of Detection (percent)	Sample Locations
Iceberg Lettuce	FDA (2004)[a]	µg/kg FW	38	1	<MRL – 71.6	7.76	79%[b]	AZ, CA, FL, NJ
	Sanchez *et al.* (2005a)[c]	µg/kg FW	44	~20	<MRL - 26	NA	86%	AZ, CA
	Sanchez *et al.* (2005a)[f]	µg/kg FW	24	25-30	ND - 24	10	NA	AZ, CA
	Sanchez *et al.* (2005b)[f]	µg/kg FW	63	20-40	ND - 31	7.4	NA	See note[m]
Romaine Lettuce	FDA (2004)[a]	µg/kg FW	40	1	<MRL - 129	11.9	95%[b]	AZ, CA, FL, NJ, TX
	Sanchez (2004)[e]	µg/kg FW	7	20 - 50	<MRL - 81	NA	100%	AZ, CA
	Sanchez *et al.* (2005a)[d]	µg/kg FW	24	25-30	ND - 20	13	NA	AZ, CA
	Sanchez *et al.* (2005b)[e]	µg/kg FW	84	20-40	ND - 100	17.1	NA	See note[m]
Green Leaf Lettuce	FDA (2004)[a]	µg/kg FW	25	1	1.00 – 27.4	10.7	100%	AZ, CA, NJ, TX
	Sanchez (2004)[e]	µg/kg FW	3	20 - 50	46-64	NA	100%	AZ, CA
	Sanchez *et al.* (2005a)[e]	µg/kg FW	24	25-30	ND - 102	33	NA	AZ, CA
	Sanchez *et al.* (2005b)[e]	µg/kg FW	69	20-40	ND - 195	16.5	NA	See note[m]
Red Leaf Lettuce	FDA (2004)[a]	µg/kg FW	25	1	<MRL – 52.0	11.6	92%[b]	AZ, CA, TX
	Sanchez *et al.* (2005a)[e]	µg/kg FW	24	25-30	ND - 81	27	NA	AZ, CA
	Sanchez *et al.* (2005b)[e]	µg/kg FW	67	20-40	ND - 104	14.5	NA	See note[m]
Butterhead Lettuce	Sanchez *et al.* (2005a)[e]	µg/kg FW	24	25-30	ND - 104	29	NA	AZ, CA
	Sanchez *et al.* (2005b)[e]	µg/kg FW	45	20-40	ND - 98	17.2	NA	See note[m]
Arugula	Sanchez *et al.* (2005b)[e]	µg/kg FW	9	20-40	ND - 195	55.8	NA	See note[m]
Spinach	Sanchez *et al.* (2005b)[e]	µg/kg FW	10	20-40	ND - 628	85.1	NA	See note[m]

Food Item	Data Reference	Units	N	MRL	Range of Detections	Reported Means[s]	Rate of Detection (percent)	Sample Locations
Bottled Water	FDA (2004)	µg/L	51	0.5	<MRL – 0.56	NA	4%[b]	CA, CO, GA, MD, MN, MO, NC, NE, PA, SC, TX, WI
Dairy Milk	FDA (2004)	µg/L	104	3	<MRL – 11.3	5.76	97%[b]	AZ, CA, GA, KS, LA, MD, MO, NJ, NC, PA, SC, TX, VA, WA
	Kirk *et al.* (2005)	µg/L	47	~1[g]	ND – 11.0	2.0	98%	AK, AZ, CA, FL, HI, KS, ME, NH, NM, NY, PA
	Kirk *et al.* (2003)	µg/L	7	0.5[g]	1.7 – 6.4	NA	100%	TX
Melon	Sanchez (2004)[h]	µg/kg FW	25	20 - 50	ND - <MRL	NA	48%	AZ, CA
	Jackson *et al.* (2005)[i]	µg/kg FW	1	NA	1600	NA	100%	KS
Cucumber	Jackson *et al.* (2005)[n]	µg/kg FW	2	NA	40 - 770	NA	100%	TX, KS
Tomato	Sanchez (2004)	µg/kg FW	8	20 - 50	ND - <MRL	NA	37%	AZ, CA
	Jackson *et al.* (2005)	µg/kg FW	2	NA	42 - 220	NA	100%	KS
Pepper	Sanchez (2004)	µg/kg FW	10	20 - 50	ND - <MRL	NA	30%	AZ, CA
Carrot	Sanchez (2004)	µg/kg FW	10	20 - 50	ND	NA	0%	CA
Onion	Sanchez (2004)	µg/kg FW	10	20 - 50	ND	NA	0%	CA
Sweet Corn	Sanchez (2004)	µg/kg FW	18	20 - 50	ND	NA	0%	AZ, CA

Food Item	Data Reference	Units	N	MRL	Range of Detections	Reported Mean[s]	Rate of Detection (percent)	Sample Locations
Squash	Sanchez (2004)	µg/kg FW	10	20 - 50	ND	NA	0%	AZ, CA
Wheat	Sanchez (2004)[j]	µg/kg FW	NA	20 - 50	ND	NA	0%	AZ
	Jackson *et al.* (2005)[k]	µg/kg FW	12	NA	710 – 4400[l]	NA	100%	TX
Alfalfa	Sanchez (2004)[o]	µg/kg FW	10	20 - 50	109 - 668	NA	100%	AZ, CA
	Jackson *et al.* (2005)[p]	µg/kg FW	3	NA	NA	2900	100%	TX
Soy Milk	Kirk *et al.* (2005)	µg/L	1	~1[g]	0.7	NA	100%	TX
Lemon	Sanchez *et al.* (2006)	µg/kg FW	33	~2.5	ND – 14.8	2.3	NA	AZ, CA
Grapefruit	Sanchez *et al.* (2006)	µg/kg FW	15	~2.5	ND – 16.2	3.3	NA	AZ, CA
Orange	Sanchez *et al.* (2006)	µg/kg FW	28	~2.5	ND – 37.6	7.4	NA	AZ, CA
Seaweed	Martinelango *et al.* (2006a)[q]	µg/kg DW	13	NA	29 - 878	NA	100%	Atlantic Ocean (ME)
Beer	Aribi *et al.* (2006)	µg/L	144	NA	0.005 – 21.096	NA	100%	47 countries (including USA)
	Aribi *et al.* (2006)	µg/L	8	NA	0.364 – 2.014	0.662[r]	100%	USA
Wine	Aribi *et al.* (2006)	µg/L	77	NA	0.029 – 50.25	NA	100%	22 countries (including USA)
	Aribi *et al.* (2006)	µg/L	12	NA	0.197 – 4.593	2.09[r]	100%	USA

Notes:
N = number of samples; MRL = minimum reporting limit; ND = not detected; FW = fresh weight; DW = dry weight; NA = not available from (or not appropriate for) the cited study.
[a] Outermost leaves of each lettuce head were removed prior to sample analysis.
[b] Rate of detection is based on number of samples for which perchlorate was quantifiable (not just detectable).

[c] Samples are of "edible head" (trimmed of frame and wrapper leaves).
[d] Samples are "bulk" (partial removal of stem core and partial severing of upper and outer leaf blade margins).
[e] Samples preparation included minimal trimming.
[f] Samples have had multiple layers of their outer wrapper leaves removed.
[g] Value reported as the "limit of detection."
[h] Samples include cantaloupe, casaba, honey dew, galia, and watermelon.
[i] Sample of cantaloupe from a home garden in Morris County, KS.
[j] Durum wheat.
[k] Whole wheat head, including seed (endosperm), bran, germ, and chaff.
[l] Represents the range of average values (3 samples, each) of 4 commercial growing fields in Gaines County, TX. In partitioned samples, perchlorate in the whole grain (not including the chaff) measured 1300 µg/kg FW in 1 sample and was not detected in 2 samples of wheat endosperm.
[m] Study was restricted to foods outside the lower Colorado River region. Sample locations were not presented for each food item, however, the complete list of regions sampled is CA, CO, MI, NJ, NM, NY, OH, and Quebec.
[n] Samples were collected from home gardens in Gaines County, TX, and Morris County, KS.
[o] Six of the ten alfalfa samples were sent to FDA for confirmatory analysis by ion chromatography-tandem mass spectrometry (IC-MS/MS). The FDA results ranged from 121 to 382 µg/kg FW.
[p] Samples were collected from a single commercial growing field in Gaines County, TX.
[q] Samples of 11 different commercially available species were collected.
[r] Value provided is the median (not the mean).
[s] When comparing means from the studies it is important to note that the different studies likely treated non-detects differently. Some studies treated non-detects as one-half the MRL and others treated non-detects as zero.

FDA Targeted Sampling

The FDA released data on perchlorate in milk, lettuce, and bottled water in November 2004. To analyze food samples, FDA used ion chromatography (IC)-tandem mass spectrometry (MS/MS), referred to as IC-MS/MS. The quantitation limits for perchlorate in these analyses were 0.5 µg/L for bottled water, 1 µg/kg by fresh weight (FW) for lettuce, and 3 µg/L for dairy milk. The mean concentration of perchlorate in 128 lettuce samples collected in 5 states (AZ, CA, FL, NJ, TX) was 10.3 µg/kg FW (FDA, 2004), and ranged from not quantifiable (NQ) to 129 µg/kg FW. The mean concentrations of perchlorate in several varieties of lettuce are reported in Exhibit 12-2. The mean concentration of perchlorate in 104 dairy milk samples collected in 14 states (AZ, CA, GA, KS, LA, MD, MO, NJ, NC, PA, SC, TX, VA, WA) was 5.76 µg/L (FDA, 2004), with a range from NQ to 11.3 µg/L. FDA (2004) detected perchlorate in 2 of the 51 bottled water samples representing 34 distinct sources collected in 12 states (CA, CO, GA, MD, MN, MO, NC, NE, PA, SC, TX, WI) at levels of 0.56 µg/L and 0.45 µg/L.

Other Published Studies

Sanchez (2004) and Sanchez *et al.* (2005a) report the results of an analysis of agricultural products sampled from the lower Colorado River region of Arizona and California, the Imperial Valley of California, and the Coachella Valley of California, where irrigation water is known or suspected to contain perchlorate. The studies were partially supported by the U.S. Department of Agriculture – Agricultural Research Service (USDA-ARS). Samples of iceberg, romaine, and leaf lettuce, carrots, onions, sweet corn, squash, melons, tomatoes, peppers, broccoli, cauliflower, cabbage, durum wheat, and alfalfa were analyzed for perchlorate using IC as the primary analytical method. For these analyses, the fresh-weight method reporting limit was not identified in most cases, but was reported to range from 20 to 50 µg/kg FW, depending on the moisture content of the samples (Sanchez, 2004). Sanchez *et al.* (2005a) report that the method reporting level for iceberg lettuce was approximately 20 µg/kg FW and for other types of lettuce was 25-30 µg/kg FW. Perchlorate in the irrigation water ranged from 1.5 to 8.0 µg/L over the period of the survey (Sanchez *et al*., 2005a).

Sanchez *et al*. (2005a) analyzed 44 samples of iceberg lettuce heads that had been trimmed of frame and wrapper leaves, which are usually removed before the lettuce is consumed. Perchlorate was quantified in 5 of the samples (ranging from 23 to 26 µg/kg FW)[5], perchlorate was not detectable in 6 samples, and the results of the remaining samples were less than the method reporting limit, which the authors defined as "a detectable peak among duplicates and/or replicates but below a level that can be quantitated." Perchlorate concentrations in 10 samples of romaine and green leaf lettuce ranged from less than the method reporting limit to 81µg/kg FW (Sanchez, 2004).

As shown in Exhibit 12-2, Sanchez (2004) also detected perchlorate in samples of melons, tomatoes, and peppers, but at levels below the method reporting limit. Perchlorate was not detected in carrots, onions, sweet corn, squash, and durum wheat. Concentrations of

5 Sanchez (2004) presents somewhat different results. Specifically, of the 44 samples of "edible head" lettuce, perchlorate was quantified in one of the samples (26 µg /kg), perchlorate was not detectable in 6 samples, and the remaining sampling results were qualified as <MRL, which the author defined as "represents a seemingly detectable peak but below a level that can be quantitated."

perchlorate in 10 samples of alfalfa ranged from 109 to 668 µg/kg FW. Six of the 10 alfalfa samples were sent to FDA for confirmatory analysis by IC-MS/MS. The FDA results were generally lower than those of the corresponding samples by Sanchez (2004), ranging from 121 to 382 µg/kg FW.

Sanchez *et al.* (2006) conducted studies to evaluate the uptake and distribution of perchlorate in citrus trees and the occurrence of perchlorate in lemons, grapefruit, and oranges grown in southern California and southwestern Arizona. Five whole lemon trees irrigated with Colorado River water were harvested for destructive sampling. Sanchez *et al.* (2006) estimate that the irrigation water had an average perchlorate concentration of 6 µg/L. Most of the sample analysis was conducted using IC-MS/MS, having an MRL of approximately 25 µg/kg by dry weight (DW). In samples of tree trunks, roots, and branches, perchlorate was close to or below the MRL. Perchlorate was much higher in the leaves than the fruit (peel and pulp), with mean concentrations of 1,835 and 128 µg/kg DW, respectively.

Citrus samples were collected during 2004-2005 from the lower Colorado River Valley, the University of Arizona Research Farm, the Coachella Valley, and Los Angeles County. All analyses of fruit pulp were conducted using IC-MS/MS with an approximate MRL of 2.5 µg/kg FW. For the 86 citrus samples collected, the perchlorate concentration in the fruit pulp ranged from below detection to 37.6 µg/kg FW. Mean concentrations in lemons (33 samples), grapefruit (15 samples), and oranges (28 samples) were 2.3, 3.3, and 7.4 µg/kg FW, respectively.

Sanchez *et al.* (2005b) surveyed perchlorate occurrence in lettuce and other leafy vegetables produced outside the lower Colorado River region. Samples were analyzed by IC, with a minimum reporting level of approximately 20 to 40 µg/kg FW, depending on the leafy vegetable type. Results of some of the more heavily sampled food items are presented in Exhibit 12-2.

While not shown in Exhibit 12-2, Sanchez *et al.* (2005b) performed additional analysis by partitioning the leafy vegetable samples by type of culture. Perchlorate was detected in 70 of 268 samples of conventionally-grown leafy vegetables and 72 of 170 samples of organically-grown leafy vegetables. The range of perchlorate concentrations was not detected (ND) to 104 µg/kg FW in conventional leafy vegetables and ND to 628 µg/kg FW in organic leafy vegetables. Sanchez *et al.* (2005b) analyzed the results using regression analysis and estimated that the median perchlorate concentration in organically-grown samples was 2.2 times higher than in conventionally-grown samples. The regression analysis also suggested that variation among sampling locations was greater than variation among lettuce types.

Researchers at Texas Tech University analyzed samples of dairy and soy milk using IC and/or IC/MS analytical methods with detection limits of 1 µg/L or better (Kirk *et al.*, 2005). In a study of perchlorate in dairy milk, Kirk *et al.* (2005) found mean perchlorate levels of 2.0 µg/L in 47 retail dairy milk samples from 11 states (AK, AZ, CA, FL, HI, KS, ME, NH, NM, NY, PA), with a range from not ND to 11.0 µg/L. A single sample of soy milk was analyzed and reported to contain 0.7 µg/L perchlorate (Kirk *et al.*, 2005). An earlier study by Kirk *et al.* (2003) found perchlorate ranging from 1.7 µg/L to 6.4 µg/L in 7 dairy milk samples purchased in a city in Texas.

Jackson *et al.* (2005) conducted limited sampling of edible and forage vegetation in 1 Texas county and in 1 Kansas home garden. In Texas, wheat and alfalfa were sampled from commercial fields irrigated with groundwater containing perchlorate from an unknown source, and a cucumber was sampled from an irrigated home garden. In Kansas, cantaloupe, cucumber, and tomatoes were sampled from an irrigated home garden near a slurry explosives site. Researchers used IC for sample analysis but did not report fresh-weight detection limits. Perchlorate was detected in all 12 samples of winter wheat heads (whole, including the chaff) at a mean concentration of 2,000 µg/kg FW but perchlorate was not detected in wheat endosperm (2 samples)[6]. The mean perchlorate concentration in 3 samples of alfalfa was 2,900 µg/kg FW. A cucumber sample from a Texas home garden contained 40 µg/kg FW perchlorate; a sample of irrigation water from this garden contained 20.7 µg/L perchlorate. In the Kansas home garden, the cucumber sample contained 770 µg/kg FW perchlorate, the cantaloupe sample contained 1,600 µg/kg FW perchlorate, and 2 samples of tomato contained 42 and 220 µg/kg FW perchlorate. The reported concentration of perchlorate in irrigation water for the Kansas home garden was 81 µg/L. EPA notes that the perchlorate levels in irrigation water samples associated with these two home gardens were significantly higher than in the vast majority of surface and ground water samples in the US.

Aribi *et al*. (2006) developed an analytical method for perchlorate that uses ion chromatography with suppressed conductivity and electrospray ionization tandem mass spectrometry (IC-ESI-MS/MS). The method was used to measure perchlorate in samples of various food products, including fresh/canned fruits and vegetables, wine, beer, and other beverages. Most samples were purchased in grocery and liquor stores in greater Toronto, Canada, between January 2005 and February 2006. Produce samples originated from many different parts of the world and all samples contained measurable amounts of perchlorate. However, the survey was limited to only a few samples of each food. Products from California, Chile, Costa Rica, Guatemala, and Mexico had the highest levels of perchlorate. Products from Canada and China had the lowest levels of perchlorate. The highest detection was in cantaloupe from Guatemala (463.50 µg/kg FW). Analysis of raw asparagus (39.900 µg/kg FW) and cooked asparagus (24.345 µg/kg FW) demonstrated that perchlorate can remain in food processed at a high temperature. Perchlorate concentrations in 8 samples of produce from the U.S. ranged from 0.094 µg/kg FW (for blueberries) to 19.29 µg/kg FW (for green grapes).

Aribi *et al.* (2006) analyzed 77 samples of wine and 144 samples of beer from many parts of the world. All samples contained measurable amounts of perchlorate. The wine sample with the single highest concentration of perchlorate, 50.250 µg/L, was from Portugal. Overall, wine samples from Chile contained the highest concentrations of perchlorate, ranging from 5.358 to 38.88 µg/L in 8 samples. Twelve samples of wine from the U.S. contained perchlorate concentrations ranging from 0.197 to 4.593 µg/L. Results from analysis of beer samples varied substantially among countries, with an overall range from 0.005 µg/L (Ireland) to 21.096 µg/L (France). Concentrations of perchlorate in 8 beer samples from the U.S. ranged from 0.364 to 2.014 µg/L.

6 A wheat kernel (seed) has three major parts - the bran, the germ, and the endosperm. The majority of the wheat kernel is the endosperm, which is the portion of the kernel that is retained in refined (white) wheat flours. Whole wheat flours contain endosperm, wheat bran, and wheat germ in approximately the same proportions as in the wheat kernel. Wheat flours do not contain the chaff (husk).

Snyder *et al.* (2006) measured perchlorate in dietary supplements and flavor enhancing ingredients collected from various vendors in Las Vegas, NV, and Seattle, WA. Analyses were performed using liquid chromatography tandem mass spectrometry (LC-MS/MS) with a limit of detection between 2 and 5 µg/kg. Perchlorate was detected in 20 of 31 analyzed supplements, with detectable concentrations ranging from 10 to 2,420 µg/kg. Based on manufacturers' recommended intake of the supplements, the resulting daily oral doses of perchlorate would range from 0.03 to 18 µg/day. Twelve of the supplements tested were prenatal or children's vitamins. The highest level of perchlorate (2,420 µg/kg or 0.018 mg/day at the recommended daily dose) was found in a prenatal vitamin; in the remaining prenatal and children's vitamins perchlorate did not exceed 28 µg/kg. The study noted that "vitamin and mineral supplements are typically formulated to include the Recommended Daily Allowance (RDA) of iodine, a factor that would provide protection against any possible impacts of microgram levels of perchlorate found in these supplements." Perchlorate was also detected at 740 µg/kg in a sample of kelp granules (a flavor enhancer), which equates to 2.2 µg perchlorate per serving.

Martinelango *et al.* (2006a) measured perchlorate in seaweed, which is often used as a source of iodide in food and nutritional supplements. Martinelango *et al.* (2006a) collected samples of 11 different species of seaweed growing off the coast of northeastern Maine. Perchlorate was detected in all species, with concentrations ranging from 29 to 878 µg/kg DW. The iodide content in the samples was much higher, ranging from 16 to 3,134 mg/kg DW. Martinelango *et al.* (2006a) found that samples of *Laminaria* species concentrated iodide more selectively than perchlorate. Laminaria is a genus of large brown seaweeds that are commonly used in kelp tablets. Martinelango *et al.* (2006a) also analyzed 4 seaweed samples that had been washed with deionized water and found that a single wash removed 38 to 73 percent of the perchlorate and 34 to 44 percent of the iodide.

12.3.5 Occurrence Studies on Perchlorate in Human Urine, Breast Milk, and Amniotic Fluid

Recently researchers have used the results of the analysis of urine samples to estimate human exposure to perchlorate. Ingested perchlorate is not metabolized by humans and is excreted largely in the urine (Merrill *et al.*, 2005). The CDC's National Center for Environmental Health (NCEH) developed a sensitive and selective analytical method to analyze perchlorate in human urine (Valentín-Blasini *et al.*, 2005). The method uses ion chromatography coupled with electrospray ionization tandem mass spectrometry (IC-ESI-MS/MS) and achieves an MRL of 0.025 µg/L in human urine. The authors report that the method is robust enough to process first-morning-void urine samples, which are samples of the first voiding of urine upon waking.

Valentín-Blasini *et al.* (2005) analyzed urine samples from 61 healthy adult donors who lived in the area of Atlanta, Georgia. The urine samples were provided anonymously, without associated donor information. Perchlorate was detected in all of the urine samples, with concentrations ranging from 0.66 to 21 µg/L. The authors cited dietary exposure as a potential source of perchlorate because perchlorate was found only at low levels (0.1 – 0.2 µg/L) in area tap water samples (Valentín-Blasini *et al.*, 2005).

Valentín-Blasini *et al.* (2005) also analyzed the urine samples for creatinine, which is a metabolic breakdown product in muscles that is eliminated from the body in urine at a predictable rate. When adjusted for urinary creatinine content, the reported range of perchlorate in the samples is 1.0 to 35 µg of perchlorate per gram of creatinine. The median perchlorate concentration was 3.2 µg/L (7.8 µg/g creatinine). The researchers stated that only 1 sample from the Atlanta population contained perchlorate at a level slightly in excess of the amount expected to be excreted by an individual exposed to perchlorate at the reference dose of 0.0007 mg/kg/day (Valentín-Blasini *et al.*, 2005). Specifically, assuming that perchlorate is excreted uniformly in urine throughout the day, a urinary excretion level of 34 µg perchlorate per gram creatinine would be associated with a daily perchlorate intake of 0.0007 mg/kg/day, for a 70 kg male that excretes creatinine at a typical rate of 1.44 grams per day (g/day). These assumptions are imprecise for individual exposure assessment but allow for spot urine perchlorate excretion to be related to the reference dose for toxicological perspective. Estimating perchlorate exposure from a single spot urine sample (as opposed to a sample collected continuously over a period of time) is imprecise due to the episodic nature of perchlorate exposure and the short half-life of perchlorate in the human body. The precision of estimated individual perchlorate exposure can be improved by more precise estimation of 24-hour creatinine excretion based on sex, height, weight, and age as described by Mage *et al.* (2004). In addition, imprecision stemming from the episodic nature of perchlorate exposure can be reduced with increased sampling.

The analytical method developed by Valentín-Blasini *et al.* (2005) was further used by Blount *et al.* (2006a) to evaluate urine samples from 27 volunteers with differing dietary habits. Blount *et al.* (2006a) collected first-morning-void urine specimens from volunteers living in the Atlanta area. The study volunteers self-assessed their consumption of milk, dairy products, and green/leafy vegetables within the 16 hours before the sample was collected. The samples were grouped into 2 categories ("one or fewer servings" and "three or more servings") based on total consumption of these selected foods. Total daily perchlorate exposure was calculated using a bodyweight of 70 kg and a creatinine excretion rate of 1.44 g/day, assuming that each first-morning void urine sample was representative of that individual's daily perchlorate exposure. Each volunteer also collected a drinking water sample from home and work. Blount *et al.* (2006a) analyzed drinking water samples with the same method used for urine analysis and estimated exposure from drinking water based on a body weight of 70 kg and daily consumption of 2 liters of water per day. The mean creatinine-adjusted urinary perchlorate level was 1.8 times higher for individuals who identified themselves as consuming three or more servings of milk, dairy products, and/or green/leafy vegetables (6.13 versus 3.45 µg/g creatinine). There were no significant differences in the perchlorate levels in the drinking water samples of the 2 diet groups, which ranged from <0.05 to 0.25 µg/L with a median of 0.10 µg/L. Using a median drinking water level of 0.10 µg/L, Blount *et al.* (2006a) estimated that the perchlorate dose from drinking water was 0.003 µg/kg/day. Compared to this drinking water estimate, the total perchlorate dose estimate based on mean urinary perchlorate excretion was 24 times higher (0.071 µg/kg/day) and 42 times higher (0.126 µg/kg/day) for the low-consumption and high-consumption diet groups, respectively. The overall range of perchlorate found in urine was 0.94 to 17 µg/g creatinine with a median of 4.2 µg/g creatinine.

In the largest study of its kind, Blount *et al.* (2006c) measured perchlorate in urine samples collected from a nationally representative sample of 2,820 U.S. residents, ages 6 years and older, as part of the 2001-2002 NHANES. Blount *et al*. (2006c) detected perchlorate at

concentrations greater than 0.05 µg/L in all 2,820 urine samples tested, with a median concentration of 3.6 µg/L (3.38 µg/g creatinine) and a 95th percentile of 14 µg/L (12.7 µg/g creatinine). Only 0.7 % of the study participants had an estimated perchlorate dose in excess of 0.0007 mg/kg/day. Women of reproductive age (15-44 years) had a median urinary perchlorate concentration of 2.9 µg/L (2.97 µg/g creatinine) and a 95th percentile of 13 µg/L (12.1 µg/g creatinine). The demographic with the highest concentration of urinary perchlorate was children (6-11 years), who had a median urinary perchlorate concentration of 5.2 µg/L (5.79 µg/g creatinine). Blount *et al.* (2006c) estimated a total daily perchlorate dose for each adult and found a median dose of 0.066 µg/kg/day (about one tenth of the RfD) and a 95th percentile of 0.234 µg/kg/day (about one third of the RfD). Eleven adults (0.7%) had estimated perchlorate exposure in excess of the RfD (0.7 µg/kg/day). The highest estimated exposure was 3.78 µg/kg/day. Because of daily variability in diet and perchlorate exposure, and the short residence time of perchlorate in the body, these single sample measurements may overestimate long-term average exposure for individuals at the upper end of the distribution and may underestimate the long term average exposure for individuals at the lower end of the distribution. Daily perchlorate dose is not presented for children and adolescents due to the limited validation of formulas for these age groups (Blount *et al.*, 2006c).

Valentín-Blasini *et al.* (2005) and Téllez *et al*. (2005) analyzed urine samples of pregnant women in 3 cities in Chile and found higher median levels of urinary perchlorate in cities with higher concentrations of perchlorate in tap water. Based on an assessment of drinking water intake, the researchers determined that, in all 3 cities, there was an additional source of perchlorate for the study participants that may be explained by dietary (food) intake (Téllez *et al*., 2005). This gap between estimated perchlorate exposure and perchlorate intake from tap water consumption ranged from 21.7 µg/day to 33.8 µg/day in the three Chilean cities (Téllez *et al*., 2005).

Martinelango *et al.* (2006b) developed a method to measure perchlorate in human urine with a limit of detection of 0.080 µg/L, and reported analytical results of 9 spot urine samples from male and female volunteers. Perchlorate was present in all samples analyzed, at concentrations ranging from 2.2 to 14.9 µg/L, with a median value of 8.1 µg/L.

Other studies have investigated perchlorate in human breast milk. Kirk *et al.* (2005) analyzed 36 breast milk samples from 18 states (CA, CT, FL, GA, HI, MD, ME, MI, MO, NC, NE, NJ, NM, NY, TX, VA, WA, WV) and found perchlorate concentrations in all samples ranging from 1.4 to 92.2 µg/L in all samples, with a mean concentration of 10.5 µg/L. Téllez *et al.* (2005) report maternal parameters for participants from the study in Chile. Breast milk samples indicated that a significant amount of perchlorate leaves the body of the nursing mother through breast milk, in addition to urine. However, the breast milk perchlorate levels were highly variable and no significant correlations could be established between breast milk perchlorate and either urine perchlorate or breast milk iodide concentrations for the individuals evaluated in these Chilean cities (Téllez *et al.*, 2005). Kirk *et al*. (2006) evaluated variations of iodide, thiocyanate and perchlorate in human milk samples. These authors suggest that if the overall intake of iodide is sufficient, it is unlikely that milk with an occasional low iodide or high perchlorate content would pose a major risk to infants. However, their limited data (evaluating only 10 women) show that the milk of some women may not supply infants with adequate iodide and they suggest that it may be important to base risk assessments for perchlorate exposure on

the iodide to perchlorate ratio or the ratio of iodide to a "selectively-weighted sum of iodide uptake inhibiting agents."

Blount and Valentín-Blasini (2006) developed a sensitive and selective method for quantifying iodide, perchlorate, thiocyanate, and nitrate in human amniotic fluid. The analytical limit of detection for perchlorate was calculated to be 0.020 µg/L. Samples of amniotic fluid at 15 to 20 weeks gestation were collected from 48 healthy women in an Eastern U.S. city for analysis. Perchlorate was found in all samples tested and exhibited a log-normal distribution. The perchlorate concentrations ranged from 0.057 to 0.71 µg/L with a median value of 0.18 µg/L.

12.4 Status of the Preliminary Regulatory Determination

At this time, the Agency is not making a preliminary regulatory determination for perchlorate. The Agency believes that additional information is needed on the sources of human exposure if it decides to base its determination regarding health risk reduction potential on a health reference level (HRL) derived from the RfD and the relative source contribution (RSC) for drinking water. Under this approach, the Agency would use the RfD and RSC to estimate an HRL and then use this HRL as a benchmark against which to conduct an evaluation of the occurrence data. In conducting such an assessment for the 6 non-carcinogens undergoing regulatory determination at this time, EPA used a 20 percent RSC, which is the lowest and most conservative RSC used to estimate an HRL. Since the initial screening of the occurrence data against the HRL resulted in a preliminary negative determination, the Agency found that it was not necessary to further evaluate the RSC for these contaminants. In the case of perchlorate, the Agency is not at the point of being able to make either a negative or a positive determination using this approach because it is not yet clear what an appropriate RSC for perchlorate is. If EPA were to use a default RSC of 20% for perchlorate, the resulting HRL would be 5 µg/L. Approximately 3.16% of the 3,858 PWSs in the UCMR 1 data set had at least one detect of perchlorate greater than or equal to 5 µg/L. Given this level of occurrence at the default-derived HRL, the Agency believes a better informed RSC and HRL would be needed to use this approach to determine whether regulation of perchlorate in drinking water presents a meaningful opportunity for health risk reduction.

Exhibit 12-3 shows the number of systems and population served that would exceed the HRL under various RSC scenarios and the sensitivity of this estimate to relatively small changes in the estimated RSC. For example, increasing the RSC from 20 to 30 percent would lower the estimated number of systems impacted by about a third, and the estimated population served by about half. Hence, the choice of an appropriate RSC and resulting HRL could impact EPA's determination of whether regulation of perchlorate represents a meaningful opportunity for health risk reduction if it uses this approach.

EPA recognizes that system-level population estimates shown in Exhibit 12-3 may be conservative because some systems have multiple entry points to the distribution system and not all entry points had a positive detection for perchlorate in the UCMR 1 survey. Hence, to derive a less conservative population estimate (last column in Exhibit 12-3), EPA assumed that the population for each system is equally distributed over all of the entry (or sampling) points and

estimated a population-served value based on entry points that had at least 1 analytical detection for perchlorate at levels greater than each of the HRL thresholds.

Exhibit 12-3: UCMR 1 Occurrence and Population Estimates for Perchlorate at Various HRL Thresholds

RSC Scenarios	Estimated HRL Thresholds Based on Various RSC Scenarios [b]	PWSs with at Least 1 Detection > Threshold of Interest	PWS Entry or Sample Points with at Least 1 Detection > Threshold of Interest [c]	Population Served by PWSs with at Least 1 Detection > Threshold of Interest [d]	Population Estimate for Entry or Sample Points Having at Least 1 Detection > Threshold of Interest [e]
20%	5 µg/L	3.16 % (122 of 3,858)	1.88 % (281 of 14,984)	14.6 M	4.0 M
30%	7 µg/L	2.13 % (82 of 3,858)	1.14 % (171 of 14,984)	7.2 M	2.2 M
40%	10 µg/L	1.35 % (52 of 3,858)	0.65 % (97 of 14,984)	5.0 M	1.5 M
50%	12 µg/L	1.09 % (42 of 3,858)	0.42 % (63 of 14,984)	3.6 M	1.2 M
60%	15 µg/L	0.80 % (31 of 3,858)	0.29 % (44 of 14,984)	2.0 M	0.9 M
70%	17 µg/L	0.70 % (27 of 3,858)	0.24 % (36 of 14,984)	1.9 M	0.8 M
80%	20 µg/L	0.49 % (19 of 3,858)	0.16 % (24 of 14,984)	1.5 M	0.7 M
100%	25µg/L	0.36 % (14 of 3,858)	0.12 % (18 of 14,984)	1.0 M	0.4 M

Footnotes:
a. These data represent summary statistics for the 3,858 public water systems that have sampled for perchlorate as a part of the UCMR 1 survey.
b. HRL threshold = [(RfD of 0.0007 mg/kg/day x 70 kg BW for pregnant female) / (2 L DWI)] x the RSC scenario. Each HRL threshold value is converted from mg/L to µg/L units and then rounded to the nearest whole number.
c. The entry/sample-point-level population served estimate is based on the system entry/sample points that had at least 1analytical detection for perchlorate greater than the HRL threshold of interest. The UCMR 1 small system survey was designed to be representative of the nation's small systems, not necessarily to be representative of small system entry points.
d. The system-level population served estimate is based on the systems that had at least 1analytical detection for perchlorate greater than the HRL threshold of interest.
e. Because the population served by each entry/sample point is not known, EPA assumed that the total population served by a particular system is equally distributed across all entry/sample points. To derive the entry/sample point-level population estimate, EPA summed the population values for the entry/sample points that had at least 1 analytical detection greater than the threshold of interest.

Exhibit 12-3 also includes information on the effects of using an RSC of 100% (that is, using an HRL set at the drinking water equivalent level or DWEL of 24.5 µg/L, rounded to a whole number). Crawford-Brown *et al*. (2006), in an estimate of risk variability from perchlorate exposure through community water systems, noted that the subjects in the original 2002 Greer *et al*. study (on which the RfD of 0.0007 mg/L was based) presumably had other sources of perchlorate exposure outside of the study and suggested that it may be appropriate to view their results as reflecting the effects of *incremental* exposure to perchlorate above the background levels already in food and water rather than the effects of *total* exposure, as is implicitly assumed when the HRL is derived using an RSC to account for other sources of exposure. Use of an RSC to derive the HRL is clearly appropriate when the RfD or cancer slope factor is derived from animal studies with carefully controlled exposure. Crawford-Brown *et al*. suggest, however, that an RSC is not necessary for perchlorate because there is no reason to assume that the background exposure of the study subjects was different than that of the general population. EPA notes that the sample size in the Greer study was small and EPA is not aware of data on their background exposure to perchlorate or how representative it may be.

While several States have recommended guidelines or public health goals for perchlorate, EPA recognizes that at least 1 State, Massachusetts[7], has already promulgated a final drinking water standard for perchlorate, that other States may set drinking water standards in the future, and that these standards could impact national occurrence estimates once these standards are fully implemented.

12.5 Potential Options for Characterizing Perchlorate Exposure and Proceeding with the Preliminary Regulatory Determination for Perchlorate

While the Agency recognizes that food and other pathways may be important sources of perchlorate exposure, the Agency believes the currently available food data (summarized in section 12.3.4) are inadequate to develop a better informed RSC (and HRL). First, some of the existing data are limited in their sample numbers, geographic coverage, and analytical method adequacy. Second, the current studies provide little or no data for several food groups (e.g., meat, poultry, fish, eggs, root and tuber vegetables, brassica vegetables, bulb vegetables, tree fruits, legumes, and cereal grains) that account for about half of the diet (by mass) for females of reproductive age (mid-teens to mid-forties).

This section presents data EPA might use to estimate an RSC based on food-borne exposure as well as on several other options that the Agency is considering to better characterize perchlorate exposure and assist the Agency in making its regulatory determination for perchlorate. These options could serve as a supplement or an alternative to developing an HRL based on a better informed RSC derived from food concentration and consumption data. Specifically, urine biomonitoring data could be used to estimate perchlorate exposure. If the Agency decides to use any of the approaches discussed in section 12.5.2, EPA will need to determine what statistics (e.g., mean, median, percentile, etc.) are most appropriate for

7 Massachusetts promulgated a final drinking water standard of 2 µg/L for perchlorate on July 28, 2006. For more information about the final standard, see http://www.mass.gov/dep/public/press/pchl0706.htm (MA DEP, 2006).

consideration in a regulatory determination. The Agency will also conduct a peer review, as appropriate, of any new methodology it decides to use.

12.5.1 Use of Food Concentration and Consumption Data to Estimate an RSC

In the past, the Agency has relied on dietary exposure information from the FDA Total Diet Study (TDS) to determine the RSC allowed for drinking water and to set health goals (i.e., Maximum Contaminant Level Goals) for several inorganic compounds (e.g., antimony, cadmium, chromium, and selenium). Under the TDS, foods are sampled at retail outlets, prepared as they would be consumed, and analyzed for a variety of analytes (e.g., nutrients, pesticides, industrial chemicals). Approximately 280 foods, covering a broad spectrum of the diet, are currently sampled in each sampling event. Sampling events (known as "market baskets") occur about 4 times per year, with each event being confined to 1 of the 4 regions of the country. The dietary intake of the analyzed compounds can be calculated for the U.S. population by multiplying the concentrations found in TDS foods by the consumption amounts for each food. FDA compiles food consumption amounts for the total U.S. population by gender and by age group[8].

FDA is including perchlorate as an analyte in the 2006 TDS. EPA believes that a comprehensive dietary intake estimate for perchlorate will be useful in evaluating dietary exposure relative to drinking water. When sufficient quantitative exposure data are available (such as the data published by FDA in conjunction with the TDS), EPA can use the procedure used previously for several regulated inorganic compounds (i.e., chromium and selenium) to calculate the relative source contribution for perchlorate. In these cases where dietary intake values were available, EPA subtracted the dietary intake value from the Drinking Water Equivalent Level DWEL and used the remainder as the allowance for water. This procedure ensures that total exposure does not exceed the RfD.

12.5.2 Use of Urinary Biomonitoring Data to Evaluate Exposure to Perchlorate

Researchers at CDC's National Center for Environmental Health (NCEH) have conducted a large national study of total perchlorate exposure through analysis of urine samples collected for NHANES 2001-2002 (Blount *et al.*, 2006b and 2006c). The use of urinary perchlorate excretion to estimate perchlorate exposure has been demonstrated in Valentín-Blasini *et al.* (2005), Téllez *et al.* (2005), and Blount *et al.* (2006c). While this would be the first time the Agency has used biomonitoring data to assist EPA in making a preliminary regulatory determination for a Contaminant Candidate List (CCL) contaminant, the Agency believes that estimating perchlorate exposure among large populations using urinary perchlorate excretion data may be appropriate for the following reasons:

- Perchlorate is not metabolized in the body and is excreted unchanged primarily via the renal pathway (Merrill *et al*., 2005),
- Perchlorate does not bioaccumulate, that is, it is excreted essentially completely (Merrill *et al*., 2005),

8 Information about FDA's TDS design, food list, analytes, and analytical results can be found at www.cfsan.fda.gov/~comm/tds-toc.html. (FDA, 2006)

- Perchlorate has a short half-life in the human body (approximately 8 hours), simplifying the estimation of daily exposure (Greer *et al.*, 2002), and
- A methodology exists that allows estimation of daily perchlorate intake from all sources (e.g., water, food) using standard creatinine adjustment factors to account for variations in urine concentration (Mage *et al.*, 2004).

The Agency could use the 2001-2002 NHANES urine data in several ways as described in the following paragraphs.

One potential approach is to use the 2001-2002 NHANES urine data to determine directly whether regulation of perchlorate in drinking water presents a meaningful opportunity for health risk reduction. More specifically, we could use the urine data (as in Blount *et al*., 2006b and c) to evaluate whether total exposure from food and water is likely to result in an appreciable risk of adverse health effects for the U.S. population. If the Agency concluded that total exposure, as estimated from the urine data, does not pose an appreciable risk, even at the upper end of the exposure distribution, then it would follow logically that reducing this exposure by regulating drinking water would not present a meaningful opportunity for health risk reduction. As summarized previously, Blount *et al.* (2006c) estimated a median total daily perchlorate dose for adults of 0.066 µg/kg/day (about one tenth of the RfD) and a 95th percentile dose of 0.234 µg/kg/day (about one third of the RfD). Only eleven adults (0.7%) had an estimated dose in excess of the RfD (0.7 µg/kg/day).

EPA could also use the 2001-2002 NHANES urine data to qualitatively evaluate the importance of the water contribution to overall exposure. For this approach, the Agency could merge data from the 2001-2002 NHANES and UCMR 1 and compare the total perchlorate exposure values (based on the urine data) for the population of individuals whose drinking water contains perchlorate at various concentration levels, ranging from non-detect to the upper end of the occurrence distribution. The intent of this analysis would be to permit the Agency to determine whether total perchlorate exposure (as measured in urine) is meaningfully correlated with concentrations in local public drinking water supplies, though EPA would only use these results qualitatively because it is not possible to match up individual urine samples with individual drinking water exposures. However, the results could be useful in determining at least qualitatively the potential significance of drinking water exposure for total exposure. If there were not a significant correlation between public water system perchlorate occurrence and individual exposure as measured through biomonitoring, this might suggest that there is not a meaningful opportunity for health risk reduction through regulation of drinking water.

The Agency could also potentially use the 2001-2002 NHANES urine data to derive an RSC to use for drinking water. This could potentially be done in several different ways as follows.

Use of Urinary Biomonitoring Total Exposure Value to Estimate an RSC

One possible approach to estimating an RSC for water would be to use the urine data to estimate total perchlorate exposure, then subtract this exposure value from the reference dose and allow the remainder as the exposure limit for water. The allowed remainder divided by the RfD would be the RSC for drinking water. This approach would yield a conservative RSC value

because the exposure used to represent food would actually correspond to both food and drinking water exposure, whereas, if it were possible to estimate the exposure from food alone, the relative amount allowed for water would be larger (resulting in a higher RSC and higher health reference value). As discussed in Section 12.3.5, above, Blount *et al.* (2006c) estimated a total daily perchlorate dose for adults from urine data and found a median dose of 0.066 µg/kg/day (about one tenth of the RfD) and a 95th percentile of 0.234 µg/kg/day (about one third of the RfD). If EPA were to use the estimated 95th percentile total dose from the Blount study as if it represented the exposure from food alone, this would suggest a residual screening-level RSC of about 70% allocated to water. One possible limitation of this approach is that the Blount study estimates exposure for adults only. Therefore, an RSC developed based upon this data would not necessarily be representative of children.

Use of the Urine Data and UCMR 1 to Deduce Exposure from Other Sources and Derive the RSC

Alternately, for those NHANES survey subjects served by public drinking water systems with positive detections for perchlorate (based on UCMR 1), EPA could estimate the expected perchlorate dose contributed by drinking water (using individual water consumption data from the NHANES survey combined with UCMR 1 data for the area in which they live) and subtract it from the total perchlorate dose (based on urinary perchlorate excretion data) to calculate the amount contributed by food. Subtraction of this calculated food contribution from the RfD would yield the amount allowed for drinking water, which could be divided by the RfD to calculate an RSC. One limitation of this methodology would be the assumption that subjects in the NHANES study are uniformly consuming drinking water that contains perchlorate at the concentration indicated in the UCMR 1 data for their area.

Use of Urinary Biomonitoring Data from Exclusive Bottled Water Drinkers to Estimate an RSC

The 2001-2002 NHANES data includes urinary perchlorate data for populations who exclusively drink bottled water. As noted in section 12.3.4, above, FDA (2004) tested 51 samples of bottled water from 34 distinct sources in 12 states and detected perchlorate in 2 samples (at levels of 0.56 µg/L and 0.45 µg/L). These levels are well below the MRL for the UCMR 1 data and would not contribute significant amounts of perchlorate relative to the RfD. If the population of exclusive bottled water drinkers is sufficiently representative of the U.S. population, these data potentially could be used to estimate the contribution of perchlorate exposure coming from food and allow the Agency to estimate an RSC for drinking water. The RSC value could be derived by subtracting the estimated perchlorate exposure for exclusive bottled water drinkers from the RfD of 0.0007 mg/kg/day, using the remainder as the allowance for drinking water. One limitation of this methodology is that the perchlorate concentration of the bottled water used by this NHANES population is not known. Hence, we would have to assume that the bottled water concentration data collected by FDA (2004) is representative of the perchlorate concentration in the bottled water used by the NHANES exclusive bottled water population. Another limitation of this approach is that it would not subtract out the fraction of the drinking water intake that comes from water used for cooking purposes (since bottled water is probably not used by most subjects in cooking and household food preparation). It would thus

produce a conservative (health protective) estimate of the RSC as it would overestimate the fraction of total exposure coming from food.

12.6 References

Aribi, H., Y.J.C. Le Blanc, S. Antonsen, T. Sakuma. 2006. Analysis of Perchlorate in Foods and Beverages by Ion Chromatography Coupled with Tandem Mass Spectrometry (IC-ESI-MS/MS). *Analytica Chimica Acta.* 567(1):39-47.

Arizona Department of Environmental Quality (ADEQ), Arizona Department of Health Services (ADHS), Arizona Department of Water Resources (ADWR), and Arizona Department of Agriculture (ADOA). 2004. *Perchlorate in Arizona: Occurrence Study of 2004*. December. Available on the Internet at: http://www.azdeq.gov/function/about/download/perch1201.pdf.

Ashford, R.D. 1994. *Ashford's Dictionary of Industrial Chemicals*. London, England: Wavelength Publications Ltd. (As cited in HSDB, 2004).

Barron, L., P.N. Nesterenko, and B. Paull. 2006 . Rapid On-line Preconcentration and Suppressed Micro-bore Ion Chromatography of Part per Trillion Levels of Perchlorate in Rainwater Samples. *Analytica Chimica Acta*. 567(1):127-134.

Blount, B.C., L. Valentín-Blasini, and D.L. Ashley. 2006a. Assessing Human Exposure to Perchlorate Using Biomonitoring. *Journal of ASTM International*. 3(7):1-6.

Blount, B.C., J.L. Pirkle, J.D. Osterloh, L. Valentín-Blasini, and K.L. Caldwell. 2006b. Urinary Perchlorate and Thyroid Hormone Levels in Adolescent and Adult Men and Women Living in the United States. *Environmental Health Perspectives*. Vol. 114, No. 12. pp. 1865-1871.

Blount, B.C., L. Valentín-Blasini, J.D. Osterloh, J.P. Mauldin, and J.L. Pirkle. 2006c. Perchlorate Exposure of the US Population, 2001–2002. *Journal of Exposure Science and Environmental Epidemiology*. Advance online publication 18 October 2006. Available on the Internet at: http://www.nature.com/jes/journal/vaop/ncurrent/pdf/7500535a.pdf.

Blount, B.C., L. Valentín-Blasini. 2006. Analysis of Perchlorate, Thiocyanate, Nitrate and Iodide in Human Amniotic Fluid using Ion Chromatography and Electrospray Tandem Mass Spectrometry. *Analytica Chimica Acta*. Vol. 567, No. 1. pp. 87-93.

Budavari, S. (ed.). 1996. *The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals*. Whitehouse Station, NJ: Merck & Co., Inc. (As cited in HSDB, 2004).

Caldwell K.L., Jones R., and Hollowell J.G. 2005. Urinary iodine concentration: United States National Health And Nutrition Examination Survey 2001–2002. *Thyroid*. Vol. 15, pp. 692–699.

CA DHS. 2006. Perchlorate in California Drinking Water: Monitoring Update. Available on the Internet at: http://www.dhs.ca.gov/ps/ddwem/chemicals/perchl/monitoringupdate.htm. Last updated June 2, 2006.

Crawford-Brown, D., B. Raucher and M. Harrod, 2006. Intersubject Variability of Risk from Perchlorate in Community Water Supplies. *Environmental Health Perspectives*. 114(7): 975-79.

Dasgupta, P.K., P. K. Martinelango, W.A. Jackson, T.A. Anderson, K. Tian, R.W. Tock, and S. Rajagopalan. 2005. The Origin of Naturally Occurring Perchlorate: The Role of Atmospheric Processes. *Environmental Science and Technology*. 39:1569-1575.

Dasgupta, P.K., J.V. Dyke, A.B. Kirk, and A.W. Jackson. 2006. Perchlorate in the United States. Analysis of Relative Source Contributions to the Food Chain. *Environmental Science and Technology*. 40(21):6608-6614.

Defense Environmental Network & Information eXchange (DENIX). 2004. Occurrence and Potential Sources of Perchlorate Releases to the Environment as of November, 2000. Available on the Internet at: https://www.denix.osd.mil/denix/Public/Library/Water/Perchlorate/occurrences.html. Accessed August 12, 2004.

Ericksen, G.E. 1983. The Chilean nitrate deposits. *American Scientist*. 71:366–374. (As cited in USEPA, 2001).

Food and Drug Administration (FDA). 2004. "Exploratory Data on Perchlorate in Food." FDA Center for Food Safety and Applied Nutrition, Office of Plant & Dairy Foods. November 2004. Available on the Internet at: http://www.cfsan.fda.gov/~dms/clo4data.html

Food and Drug Administration (FDA). 2006. Center for Food Safety and Applied Nutrition, Office of Plant & Dairy Foods. Total Diet Study. Available on the Internet at: http://www.cfsan.fda.gov/~comm/tds-toc.html.

Gerhartz, W. (exec ed.). 1985. *Ullmann's Encyclopedia of Industrial Chemistry*, 5th ed., Vol A1. Deerfield Beach: FL: VCH Publishers. (As cited in HSDB, 2004).

Greer, M.A., G. Goodman, R.C. Pleuss, and S.E. Greer. 2002. Health Effect Assessment for Environmental Perchlorate Contamination: The Dose Response for Inhibition of Thyroidal Radioiodide Uptake in Humans. *Environmental Health Perspectives*. 110:927-937.

Gullick, R., M.W. Lechevallier, and T.S. Barhorst. 2001. Occurrence of Perchlorate in Drinking Water Sources. *Journal of the American Water Works Association.* January. pp. 66-77.

Hazardous Substances Data Bank (HSDB). 2004. Search for Potassium, Sodium or Magnesium Perchlorate. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed November 1, 2004.

Jackson, W.A., K. Rainwater, T. Anderson, T. Lehman, R. Tock, S. Rajagopalan, and M. Ridley. 2004. Distribution and Potential Sources of Perchlorate in the High Plains Region of Texas: Final Report. Texas Tech University Water Resources Center. Submitted to: Texas Commission on Environmental Quality. August 31. Available on the Internet at: http://www.waterresources.ttu.edu/final%20report.pdf.

Jackson, W.A., S.K. Anandam, T. Anderson, T. Lehman, K. Rainwater, S. Rajagopalan, M. Ridley, and R. Tock. 2005. Perchlorate Occurrence in the Texas Southern High Plains Aquifer System. *Ground Water Monitoring and Remediation*. 25(1):137-149.

Kang, N., T.A. Anderson, and W.A. Jackson. 2006. Photochemical Formation of Perchlorate from Aqueous Oxychlorine Anions. *Analytica Chimica Acta*. 567(1):pp. 48-56.

Kirk, A.B., E.E. Smith, K. Tian, T.A. Anderson, and P.K. Dasgupta. 2003. Perchlorate in Milk. *Environmental Science and Technology*. 37(21):4979-4981.

Kirk, A.B., P.K. Martinelango, K. Tian, A. Dutta, E.E. Smith, and P.K. Dasgupta. 2005. Perchlorate and Iodide in Dairy and Breast Milk. *Environmental Science and Technology*. 39(7):2011-2017.

Kirk, A.B., J.V. Dyke, C.F. Martin, and P.K. Dasgupta. 2006. Temporal Patterns in Perchlorate, Thiocyanate and Iodide Excretion in Human Milk. *Environmental Health Perspectives Online.* November 2006. In press.

Lide, D.R. (ed.). 2000. *CRC Handbook of Chemistry and Physics*. 81st ed. Boca Raton, FL: CRC Press Inc. (As cited in HSDB, 2004).

Mage, D.T., R. Allen, G. Gondy, W. Smith, D.B. Barr, and L.L. Needham. 2004. Estimating Pesticide Dose from Urinary Pesticide Concentration Data by Creatinine Correction in the Third National Health and Nutrition Examination Survey (NHANES-III). *Journal of Exposure Analysis and Environmental Epidemiology.* 14:457-465.

Martinelango, P.K., K. Tian, and P.K. Dasgupta. 2006a. Perchlorate in Seawater: Bioconcentration of Iodide and Perchlorate by Various Seaweed Species. *Analytica Chimica Acta*. 567(1):100-107.

Martinelango, P.K., G. Gümüs, and P.K. Dasgupta. 2006b. Matrix Interference Free Determination of Perchlorate in Urine by Ion Association–Ion Chromatography–Mass Spectrometry. *Analytica Chimica Acta.* 567(1):79-86.

Massachusetts Department of Environmental Protection (MassDEP). 2005. *The Occurrence and Sources of Perchlorate in Massachusetts*. Draft Report. August. Available on the Internet at: http://www.mass.gov/dep/cleanup/sites/percsour.pdf.

Massachusetts Department of Environmental Protection (MA DEP). 2006. Press Release. *Massachusetts' First-In-The-Nation Perchlorate Standards Take Effect Today Drinking Water, Cleanup Standards of 2 ppb Are Set to Protect Public Health*. July 28, 2006. Available on the Internet at: http://www.mass.gov/dep/public/press/pchl0706.htm.

Merrill, E.A., R.A. Clewell, P.J. Robinson, A.M. Jarabek, J.M. Gearhart, T.R. Sterner, and J.W. Fisher. 2005. PBPK Model for Radioactive Iodide and Perchlorate Kinetics and Perchlorate-Induced Inhibition of Iodide Uptake in Humans. *Toxicological Sciences.* 83:25-43.

National Research Council (NRC). 2005. *Health Implications of Perchlorate Ingestion*. National Academies Press, Board on Environmental Studies and Toxicology. January 2005. 276 p.

New Jersey Drinking Water Quality Institute (NJDWQI). 2005. Maximum Contaminant Level Recommendation for Perchlorate. Report submitted to the State of New Jersey Department of Environmental Protection, October 7, 2005. Available on the Internet at: http://www.state.nj.us/dep/watersupply/perchlorate_mcl_10_7_05.pdf.

Orris, G.J., G.J. Harvey, D.T. Tsui, and J.E. Eldrige. 2003. Preliminary Analyses for Perchlorate in Selected Natural Materials and their Derivative Products. USGS Open-File Report 03-314. Available on the Internet at: http://geopubs.wr.usgs.gov/open-file/of03-314/OF03-314.pdf#search=%22Preliminary%20Analyses%20for%20Perchlorate%20in%20Selected%20Natural%20Materials%20and%20%22

Rajagopalan, S., T.A. Anderson, L. Fahlquist, K.A. Rainwater, M. Ridley, and W.A. Jackson. 2006. Widespread Presence of Naturally Occurring Perchlorate in High Plains of Texas and New Mexico. *Environmental Science and Technology*. 40:3156-3162.

Sanchez, C. A. 2004. *Perchlorate Residues on Agricultural Produce Irrigated by Water from the Colorado River*. Report Submitted to the USDA-ARS under a Cooperative Agreement.

Sanchez, C.A., R.I Krieger, N. Khandaker, R.C. Moore, K.C. Holts, and L.L. Neidel. 2005a. Accumulation and Perchlorate Exposure Potential of Lettuce Produced in the Lower Colorado River Region. *Journal of Agricultural and Food Chemistry*. 53:5479-5486.

Sanchez C.A., K.S. Crump, R.I. Krieger, N.R. Khandaker, and J.P. Gibbs. 2005b. Perchlorate and Nitrate in Leafy Vegetables of North America. *Environmental Science and Technology*. 39(24):9391-9397.

Sanchez C.A., R.I. Krieger, N.R. Khandaker, L. Valentín-Blasini, and B.C. Blount. 2006. Potential Perchlorate Exposure from Citrus sp. Irrigated with Contaminated Water. *Analytica Chimica Acta.* 567(1):33-38.

Schilt, A.A. 1979. *Perchloric Acid and Perchlorates*. GFS Chemicals, Inc.: Columbus, OH. p. 35. (As cited in USEPA, 2001).

Snyder, S.A., R.C. Pleus, B.J. Vanderford, and J.C. Holady. 2006. Perchlorate and Chlorate in Dietary Supplements and Flavor Enhancing Ingredients. *Analytica Chimica Acta*. 567(No. 1):26-32.

Téllez, R.T., P.M. Chacón, C.R. Abraca, B.C. Blount, C.B. Van Landingham, K.S. Crump, and J.P. Gibbs. 2005. Chronic Environmental Exposure to Perchlorate Through Drinking Water and Thyroid Function During Pregnancy and the Neonatal Period. *Thyroid*. 15(9):963-975.

USEPA. 2001. Survey *of Fertilizers and Related Materials for Perchlorate (CLO_4^-): Final Report*. EPA Report 600-R-01-047. May 2001.

USEPA. 2002. *Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization*. External Review Draft. NCEA-1-0503. Washington, DC: National Center for Environmental Assessment. Available for download from the Internet at: http://cfpub2.epa.gov/ncea/cfm/recordisplay.cfm?deid=24002. Accessed August 24, 2004.

USEPA. 2005. "Integrated Risk Information System (IRIS), Perchlorate and Perchlorate Salts." February 2005. Available on the Internet at: http://www.epa.gov/iris/subst/1007.htm. Accessed February 2, 2005.

USEPA. 2006. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-06-008. Draft. December 2006.

Valentín-Blasini, L., J.P. Mauldin, D. Maple, and B.C. Blount. 2005. Analysis of Perchlorate in Human Urine Using Ion Chromatography and Electrospray Tandem Mass Spectrometry. *Analytical Chemistry*. 77:2475-2481.

Wang, H., A. Eaton, and B. Narloch. 2002. *National Assessment of Perchlorate Contamination Occurrence.* Denver: Awwa Research Foundation and American Water Works Association. Abstract available on the Internet at: http://www.awwarf.org/research/topicsandprojects/execSum/2508.aspx. Accessed August 12, 2004.

Weast, R.C. (ed.). 1979. *Handbook of Chemistry and Physics*, 60th ed. Boca Raton, FL: CRC Press, Inc. (As cited in HSDB, 2004).

World Health Organization (WHO). 1994. *Indicators for Assessing Iodine Deficiency Disorders and Their Control through Salt Iodization*. WHO/NUT/94.6. Geneva: World Health Organization(WHO)/International Council for the Control of Iodine Deficiency Disorders.

Chapter 13: Metolachlor

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Contents

Exhibits

Abbreviations

a.i.	Active Ingredient
CAS	Chemical Abstracts Service
CCL 2	Second Contaminant Candidate List
CWS	Community Water System
CWSS	Community Water System Survey
ESA	Ethane Sulfonic Acid
HRL	Health Reference Level
MRL	Minimum Reporting Level
MTBE	Methyl tertiary-butyl ether
NAWQA	National Water Quality Assessment
NCFAP	National Center for Food and Agricultural Policy
NOAEL	No Observed Adverse Effect Level
NPS	National Pesticide Survey
OA	Oxalic Acid
PGWDB	Pesticides in Ground Water Database
PWS	Public Water System
RfD	Reference Dose
RL	Reporting Limit
SDWA	Safe Drinking Water Act
SOC	Synthetic Organic Compound
UCM	Unregulated Contaminant Monitoring
UF	Uncertainty Factor
USGS	United States Geological Survey

13 Metolachlor

13.1 Definition

Metolachlor is a synthetic organic compound (SOC) with a Chemical Abstracts Service (CAS) registry number of 51218-45-2. Metolachlor is given the following chemical name: 2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl)acetamide (USEPA, 1995). As a compound containing one chiral carbon atom, metolachlor can exist as either of a pair of enantiomers, designated R- and S-. In cases where the isomers are present in equal proportion, the mixture is referred to as racemic. Most of the information available for metolachlor pertains to the racemic mixture; however, in certain cases, enantiomer-specific information is presented. Trade names for metolachlor include Dual, Bicep, Codal, Cotoran multi, Milocep, Primagram, Primextra, Pennant, and Ontrack 8E (USEPA, 2000 as cited in HSDB, 2004).

13.1.1 Properties and Sources

Metolachlor is an odorless liquid that is clear to white in color when isolated or tan when in formulations (Budavari, 1996; Tomlin, 1997 both as cited in HSDB, 2004). Metolachlor belongs to the chloroacetanilide class of herbicides and works through the inhibition of protein synthesis. It is used on a variety of crops, including corn, soybeans, and sorghum, as well as for hedgerows and landscape plantings (USEPA, 1995). Metolachlor is often used in formulations with other pesticides (particularly herbicides) including atrazine, cyanazine, and fluometuron (Extoxnet, 1993).

Metolachlor is largely manufactured by the Monsanto Company and by the Ciba-Geigy Corporation (SRI International, 2000 as cited in HSDB, 2004; Extoxnet, 1993). It is most often produced as a wettable powder. Metolachlor is miscible with benzene, toluene, xylene, dimethylformamide, ethylene dichloride, cyclohexanone, methanol, and dichloromethane (Tomlin, 1997 as cited in HSDB, 2004). Other physical and chemical properties of metolachlor are listed in Exhibit 13-1.

Exhibit 13-1: Physical and Chemical Properties of Metolachlor

Identification	
CAS number	51218-45-2
Molecular Formula	$C_{15}H_{22}ClNO_2$
Physical and Chemical Properties	
Boiling Point	100 °C at 0.001 mm Hg [1]
Melting Point	- 62.1 °C [2]
Molecular Weight	283.80 g/mol [1]
K_{oc}	22 - 310 [3]
Log K_{ow}	3.13 [4]
Water Solubility	530 mg/L at 20 °C [5]
Vapor Pressure	3.14×10^{-5} mm Hg at 25 °C [5]
Henry's Law Constant	9.0×10^{-9} atm-m^3/mole at 20 °C [6] 3.7×10^{-7} (dimensionless), predicted [7]
Freundlich Isotherm Constant (K)	98,200 $(\mu g/g)(L/\mu g)^{1/n}$ [8]

[1] *Budavari, 1996 (as cited in HSDB, 2004)*

[2] *Tomlin, 1997 (as cited in HSDB, 2004)*

[3] *HSDB, 2004*

[4] *Hansch et al., 1995 (as cited in HSDB, 2004)*

[5] *Wauchope et al., 1992 (as cited in HSDB, 2004)*

[6] *Chesters et al., 1989 (as cited in HSDB, 2004)*

[7] *Speth et al., 2001*

[8] *Speth and Miltner, 1990 (as cited in Speth et al., 2001)*

13.1.2 Environmental Fate and Behavior

Due to the relatively low soil/water partitioning of metolachlor, the compound is expected to be moderately to highly mobile in soil. Substantial leaching of metolachlor from soil by run-off is expected to occur (USEPA, 1995). The mobility of metolachlor in soil varies depending on the characteristics of the soil where it is applied: high organic content may increase sorption (USEPA, 1995).

Based on its relatively low Henry's Law constant and vapor pressure, metolachlor is expected to be essentially nonvolatile from soil and water under most environmental conditions (Lyman *et al*., 1990 as cited in HSDB, 2004). In soil, microbial activity appears to be the primary method of degradation of chloroacetanilide herbicides such as metolachlor (Zimdahl and Clark, 1982; Potter and Carpenter, 1995 both as cited in Rheineck and Postle, 2000). Ahrens

(1994 as cited in HSDB, 2004) reports half-lives of 67-122 days from field experiments. Half-lives under aerobic and anaerobic conditions in a sandy loam soil are reported as 67 days and 81 days, respectively (USEPA, 1995).

Volatilization and photolysis of metolachlor are not expected to be significant removal mechanisms from water (Lyman *et al*., 1990 and Chesters *et al*., 1989 both as cited in HSDB, 2004). Hessler and Frimmel (1992 as cited in HSDB, 2004) found that photolysis is hindered by the presence of humic substances in water. Other studies have demonstrated an aqueous photolysis half-life of 70 days and a soil photolysis half-life of 8 days following exposure to natural sunlight (USEPA, 1995). Metolachlor is relatively resistant to hydrolysis at pH values of 5, 7, and 9, with no significant degradation observed after 30 days (USEPA, 1995). Gustafson (1989 as cited in HSDB, 2004) reports an estimated hydrolysis half-life in water of 210 days. Half-lives under aerobic and anaerobic conditions in water are reported as 47 days and 78 days, respectively (USEPA, 1995). Empirically, the half-life of metolachlor in lake water under summer conditions was reported to be 11 days (Kochany and Maguire, 1994 as cited in HSDB, 2004).

Metolachlor undergoes biodegradation in soil; five degradates have been identified (Chesters *et al.*, 1989 as cited in HSDB, 2004). The two primary degradates are metolachlor ESA (ethane sulfonic acid) and metolachlor OA (oxanilic acid). The transformation by soil microorganisms of metolachlor to its primary degradates has been suggested to occur as a result of displacement of the chlorine atom of the parent compound by glutathione, followed by the formation of the ESA and OA degradates by different enzymatic pathways (Barbash *et al.*, 1999). The ESA and OA degradates of metolachlor can be persistent in soil; Phillips *et al.* (1999a) found that the degradates persisted in agricultural soils for more than four years after application. The metabolites are also relatively mobile; Thurman *et al.* (1996 as cited in Rheineck and Postle, 2000) have attributed their mobility to their greater solubility relative to the parent compound. Due to their mobility, the metabolites may be transported into ground water and surface water, and may be detected more frequently and often at higher concentrations than the parent compounds (Kalkhoff *et al*., 1998; Rheineck and Postle, 2000; Trent and Paulsen, 2002; Phillips, *et al*., 1999a; Phillips, *et al*., 1999b; Eckhardt, *et al*., 1999). Once in ground water, the degradation products are likely to persist for long periods of time because microbial degradation in ground water appears to be limited (Potter and Carpenter, 1995 as cited in Rheineck and Postle, 2000).

13.2 Health Effects

The Agency established a reference dose (RfD) for metolachlor of 0.1 mg/kg/day based on a "no-observed-adverse-effect level" (NOAEL) of 9.7 mg/kg/day and an uncertainty factor (UF) of 100 (USEPA, 1995). The Agency derived the NOAEL from a one-year chronic feeding study in beagle dogs where the critical effect was decreased body weight gain. Metolachlor shows some evidence of causing developmental toxicity effects in rats but none in rabbits. The doses associated with the developmental effect in rats are greater than the NOAEL and therefore the NOAEL would be protective against developmental toxicity.

Metolachlor has been evaluated for carcinogenic activity in both rats and mice. No treatment-related cancer effects were observed in 2 studies using mice. In studies using rats,

metolachlor caused a significant increase in liver nodules and carcinomas in high dose females. Negative results from mutagenicity studies suggest that tumors may result from a nonmutagenic mode of action. In 1991, a peer review committee recommended that metolachlor be classified as a possible human carcinogen based on increases in liver tumors in the female rat. However, a peer review conducted in July 1994 recommended that the evidence for cancer was suggestive and should not be quantified. This recommendation was supported by negative mutagenicity data and recent metabolism data indicating that the formation of the metabolite presumed to be the ultimate carcinogen is very low (USEPA, 1995).

13.3 Occurrence and Exposure

13.3.1 Use and Environmental Release

Metolachlor, a broad spectrum herbicide, was first registered in 1976 for general weed control in noncrop areas. Registration has since been extended to include use on corn, cotton, peanuts, pod crops, potatoes, safflowers, sorghum, soybeans, stonefruits, tree nuts, non-bearing citrus, non-bearing grapes, cabbage, certain peppers, buffalograss, guymon bermudagrass for seed production, nurseries, hedgerows/fencerows, and landscape plantings. Syngenta (formerly Ciba-Geigy) is the sole producer and primary registrant of metolachlor (USEPA, 1995). Syngenta currently markets the S-isomer, under the name S-metolachlor, as the active ingredient in the product Pennant Magnum (Syngenta, 2000).

National estimates of agricultural use for metolachlor are available from several sources. Using data from the U.S. Department of Agriculture and Resources for the Future and its own proprietary data, EPA has estimated that approximately 58.7 million pounds of metolachlor active ingredient (a.i.) were applied annually between 1987 and 1993 on registered agricultural sites (USEPA, 1995).

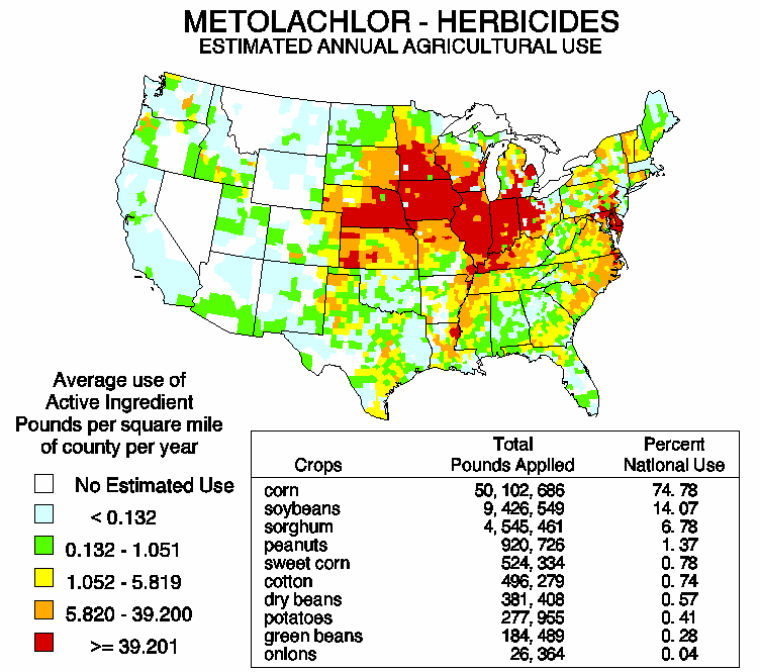
According to the National Center for Food and Agricultural Policy (NCFAP), around 1992 approximately 59.4 million pounds of metolachlor a.i. were applied annually to 16 types of crops on 32.4 million acres, and around 1997 approximately 67.3 million pounds of metolachlor a.i. were applied annually to 21 types of crops on 36.7 million acres. NCFAP estimates are based on State-level commercial agriculture usage estimates for the periods 1990-1993 and 1995-1998, and State-level estimates of crop acreage for 1992 and 1997 (NCFAP, 2004). For more information on NCFAP pesticide use estimates, see Chapter 2.

The United States Geological Survey (USGS) combined data collected by NCFAP with data from the Census of Agriculture to estimate that 57.9 million pounds of metolachlor a.i. were used annually in the early 1990s (Thelin and Gianessi, 2000). While USGS has not published national estimates for 1997, an estimate of approximately 67.0 million pounds a.i. can be inferred from the "total pounds applied" and "percent national use" data in the 1997 geographical distribution map (see below).

Exhibit 13-2 shows the estimated geographic distribution and intensity of typical annual metolachlor use in the United States in the late 1990s. A breakdown of use by crop is also included. The map was created by the USGS using State-level data sets on pesticide use rates from 1995-1998 compiled by the National Center for Food and Agricultural Policy (NCFAP),

and from county-level data on harvested crop acreage obtained from the 1997 Census of Agriculture (USGS, 2004). Due to the nature of the data sources, non-agricultural uses are not reflected here and variations in use at the county-level are also not well represented (Thelin and Gianessi, 2000). For background on the USGS pesticide use maps, see Chapter 2. The map indicates that metolachlor use is heaviest in the Midwest, but common throughout the country.

Exhibit 13-2: Estimated Annual Agricultural Use of Metolachlor, c. 1997



Crops	Total Pounds Applied	Percent National Use
corn	50, 102, 686	74. 78
soybeans	9, 426, 549	14. 07
sorghum	4, 545, 461	6. 78
peanuts	920, 726	1. 37
sweet corn	524, 334	0. 78
cotton	496, 279	0. 74
dry beans	381, 408	0. 57
potatoes	277, 955	0. 41
green beans	184, 489	0. 28
onions	26, 364	0. 04

Source: USGS, 2004

13.3.2 Ambient Water Occurrence

Ambient lakes, rivers, and aquifers are the source of most drinking water. Data on the occurrence of metolachlor in ambient surface and ground water are available from the National Water Quality Assessment (NAWQA) program of the USGS. For details on this program, see the discussion in Chapter 2. NAWQA data have been analyzed independently by USGS and EPA.

NAWQA National Pesticide Synthesis

Under the NAWQA program, USGS monitored metolachlor between 1992 and 2001 in representative watersheds and aquifers across the country. Reporting limits varied but did not exceed 0.013 µg/L.

In surface water (Exhibit 13-3), metolachlor was detected at frequencies ranging from 29.11% of samples in undeveloped areas to 49.74% of samples in urban settings, 71.37% of samples in mixed land use settings, and 82.74% of samples in agricultural areas. The 95th percentile concentrations ranged from non-detects in undeveloped areas to 1.38 µg/L in

agricultural areas. The highest maximum concentration, estimated at 77.6 µg/L, occurred in an agricultural land use setting (Martin *et al.*, 2003).

Exhibit 13-3: USGS National Synthesis Summary of NAWQA Monitoring of Metolachlor in Ambient Surface Water, 1992-2001

Land Use Type	No. of Samples (No. of Sites)	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,887 (78)	82.74%	0.029 µg/L	1.38 µg/L	77.6 µg/L (E)
Mixed	1,023 (47)	71.37%	0.010 µg/L	0.335 µg/L	9.10 µg/L
Undeveloped	60 (4)	29.11%	<RL	<RL	0.027 µg/L
Urban	885 (32)	49.74%	0.003 µg/L	0.056 µg/L	2.42 µg/L

Notes:
RL = Reporting limit. Reporting limits for metolachlor varied, but did not exceed 0.013 µg/L.

E = Estimated (outside normal calibration limits)

The USGS National Pesticide Synthesis used one year of data, generally the year with the most sampling results, to represent each site in this analysis. The sampling results were time-weighted, to eliminate bias from more frequent sampling at certain times of year. Detection Frequencies and Percentile Concentrations can be interpreted as representing annual occurrence. For instance, the detection frequency can be thought of as the percent of the year in which detections are found at a typical site in this land use category, and the 95th percentile concentration can be thought of as a concentration that is not exceeded for 95% of the year at a typical site in this land use category.

Source: Martin et al., 2003

In ground water (Exhibit 13-4), metolachlor detection frequencies ranged from 1.49% of samples in undeveloped settings to 5.04% in mixed land use settings, 8.98% in urban settings and 17.0% in agricultural settings. The 95th percentile concentrations were 0.022 µg/L in agricultural settings, and non-detects in other settings. The highest concentration, estimated at 32.8 µg/L, was found in an agricultural setting (Kolpin and Martin, 2003).

Exhibit 13-4: USGS National Synthesis Summary of NAWQA Monitoring of Metolachlor in Ambient Ground Water, 1992-2001

Land Use Type	Number of Wells	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,443	17.0%	<RL	0.022 µg/L	32.8 µg/L (E)
Mixed (Major Aquifer)	2,717	5.04%	<RL	<RL	2.62 µg/L
Undeveloped	67	1.49%	<RL	<RL	0.005 µg/L
Urban	835	8.98%	<RL	<RL	2.09 µg/L

Notes:

RL = Reporting limit. Reporting limits for metolachlor varied, but did not exceed 0.013 µg/L.

The USGS Pesticide National Synthesis considered each well a distinct site in this analysis. Each well was represented by one sample: normally the first one taken, but possibly a later sample if the first sample was not analyzed for the full range of analytes.

Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

Source: Kolpin and Martin, 2003

EPA Summary Analysis of NAWQA Data

Whereas the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination, including metolachlor. Detection frequencies were simply computed as the percentage of samples and sites with detections (i.e., with at least one result equal to or greater than the reporting limit). Note that reporting limits were not uniform. Sample detections can be biased by frequent sampling in areas with high (or low) occurrence. Calculating the percentage of sites with detections can reduce this bias. For more details on the data set and the EPA analysis, see Chapter 2.

The results of the EPA analysis are presented in Exhibit 13-5. Overall, metolachlor was detected in 53.0% of samples and at 25.4% of sites. Metolachlor was detected more frequently and at higher concentrations (maximum of 77.6 µg/L) in surface water.

Exhibit 13-5: EPA Summary Analysis of Metolachlor Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	15,634	68.9%	1,948	62.6%	0.0004	0.028	1.64	7	77.6
ground water	6,108	12.3%	5,217	11.4%	0.0002	0.007	0.364	2.43	32.8
all sites	21,742	53.0%	7,165	25.4%	0.0002	0.025	1.51	6.71	77.6

[1] RLs (Reporting Limits) for metolachlor varied but did not exceed 0.013 µg/L. For more information, see Chapter 2. Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible.

13.3.3 Drinking Water Occurrence

Nationally representative data on metolachlor occurrence in drinking water were collected by large and small public water systems in Round 2 (1993-1999) of EPA's Unregulated Contaminant Monitoring (UCM) program.

UCM Program, Round 2

Round 2 of the UCM lasted from 1993 to 1999. A geographical cross-section of States with the most complete and reliable data was chosen to provide a roughly representative picture of national occurrence in each round. Note that one of the Round 2 cross-section States with high data quality overall, Massachusetts, had data quality problems specific to metolachlor and other SOCs, and thus was not included in the cross-section analysis for metolachlor. For a complete description of the UCM program, see Chapter 2.

Exhibit 13-6 shows the results from the Round 2 cross-section (excluding Massachusetts). Results from all States, including those with incomplete and less reliable data, are also presented for the sake of comparison. Results are analyzed at the level of simple detections (at or above the minimum reporting level, or ≥ MRL--MRLs varied). Results are also analyzed at the level of a health reference level (HRL) of 70 µg/L, and at the level of ½ the HRL, or 35 µg/L.[1]

In Round 2 cross-section States, metolachlor was detected at 0.83% of public water systems (PWSs), affecting 11.58% of the population served, equivalent to approximately 24.7 million people nationally. While detections of metolachlor where primarily found in surface water systems, no detected concentration of metolachlor exceeded the HRL or ½ the HRL at any of the PWSs in the Round 2 cross-section of States.

1 The HRL is derived from the RfD by applying a risk management factor of 10 to account for suggestive evidence of carcinogenicity, and a 20-percent relative source contribution.

When all Round 2 results are included in the analysis, including results from States with incomplete or less reliable data, metolachlor occurrence findings appear to be slightly greater than those observed for the cross-section data. Detections affect 1.20% of PWSs and 14.41% of the population served. Again, no detected concentration of metolachlor exceeded the HRL or ½ the HRL.

Exhibit 13-6: Summary UCM Occurrence Statistics for Metolachlor (Round 2)

Frequency Factors	19 State Cross-Section[1]		All Reporting States[2]		National System & Population Numbers[3]	
Total Number of Samples	33,930		42,798		--	
Percent of Samples with Detections	0.57%		0.86%		--	
99th Percentile Concentration (all samples)	< MRL		< MRL		--	
Health Reference Level (HRL)	70 µg/L		70 µg/L		--	
Minimum Reporting Level (MRL) - Range - (modal value)[4]	0.01 - 52 µg/L 0.2 µg/L		0.01 - 52 µg/L 0.1 µg/L		--	
Maximum Concentration of Detections	13.8 µg/L		13.8 µg/L		--	
99th Percentile Concentration of Detections	7.1 µg/L		6 µg/L		--	
Median Concentration of Detections	0.61 µg/L		1.0 µg/L		--	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	12,953 11,503 1,450		14,878 13,062 1,816		65,030 59,440 5,590	
Total Population Population of GW PWSs Population of SW PWSs	47,098,573 14,279,627 32,818,946		59,101,488 15,749,200 43,352,288		213,008,182 85,681,696 127,326,486	
Occurrence by System	**Number**	**Percentage**	**Number**	**Percentage**	**National Extrapolation[5]**	
					Cross-Section	**All States**
PWSs with detections (≥ MRL) Range across States GW PWSs with detections SW PWSs with detections	108 0 - 40 13 95	0.83% 0 - 20.00% 0.11% 6.55%	178 0 - 60 47 131	1.20% 0 - 20.0% 0.36% 7.21%	542 N/A 67 366	778 N/A 214 403
PWSs > 1/2 HRL Range across States GW PWSs > 1/2 HRL SW PWSs > 1/2 HRL	0 0 0 0	0.00% 0 - 0.00% 0.00% 0.00%	0 0 0 0	0.00% 0 - 0.00% 0.00% 0.00%	0 N/A 0 0	0 N/A 0 0
PWSs > HRL Range across States GW PWSs > HRL SW PWSs > HRL	0 0 0 0	0.00% 0 - 0.00% 0.00% 0.00%	0 0 0 0	0.00% 0 - 0.00% 0.00% 0.00%	0 N/A 0 0	0 N/A 0 0
Occurrence by Population Served						
Population served by PWSs with detections Range across States Pop. Served by GW PWSs with detections Pop. Served by SW PWSs with detections	5,452,616 0 - 4,575,644 99,372 5,353,244	11.58% 0 - 44.41% 0.70% 16.31%	8,516,409 0 - 4,575,644 172,839 8,343,570	14.41% 0 - 48.02% 1.10% 19.25%	24,660,000 N/A 596,000 20,769,000	30,694,000 N/A 940,000 24,505,000
Population served by PWSs > 1/2 HRL Range across States Pop. Served by GW PWSs > 1/2 HRL Pop. Served by SW PWSs > 1/2 HRL	0 0 0 0	0.00% 0 - 0.00% 0.00% 0.00%	0 0 0 0	0.00% 0 - 0.00% 0.00% 0.00%	0 N/A 0 0	0 N/A 0 0
Population served by PWSs > HRL Range across States Pop. Served by GW PWSs > HRL Pop. Served by SW PWSs > HRL	0 0 0 0	0.00% 0 - 0.00% 0.00% 0.00%	0 0 0 0	0.00% 0 - 0.00% 0.00% 0.00%	0 N/A 0 0	0 N/A 0 0

1. Summary Results based on 19-State Cross-Section, UCM Round 2 data.
2. Summary Results based on All Reporting States, UCM Round 2 data.
3. Total PWS and population numbers are from EPA March 2000 Water Industry Baseline Handbook, 2nd Edition.
4. Because several different analytical methods were used, MRLs were not uniform. The modal value is the most common MRL.
5. National extrapolations are generated by multiplying the system/population percentages and the national Baseline Handbook system/population numbers.

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with Detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with Detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-Due to differences between the ratios of GW and SW systems with monitoring results and the national ratio, extrapolated GW and SW figures might not add up to extrapolated totals.
-The HRL used in this analysis is a draft value for working review only.

Each of the following maps focuses on a somewhat different aspect of the geographical distribution of metolachlor occurrence. Exhibit 13-7 identifies all States with at least one PWS with a detection of metolachlor in Round 2. All States are included in this analysis, including both cross-section States with reliable data and non-cross-section States with less reliable data, in order to provide the broadest assessment of possible metolachlor occurrence.

Exhibit 13-8 illustrates the geographic distribution of States with different detection frequencies (percentage of PWSs with at least one detection). Only cross-section States, which have the most complete and reliable occurrence data, are included in this analysis. Massachusetts, normally a Round 2 cross-section State, is excluded from the analysis due to problems with its metolachlor data.

In each map, States not analyzed are represented in white if they were not included in the relevant Round or cross-section, or the lightest category of shading if the State was included in the Round or cross-section but no data are available for metolachlor. The darker shades are used to differentiate States that have and do not have detections.

These maps reveal no clear geographic pattern of metolachlor occurrence. States with PWSs with detections are distributed from the east to the west coast, and from the Canadian to the Mexican borders.

Exhibit 13-7: Geographic Distribution of Metolachlor Detections in Both Cross-Section and Non-Cross-Section States (UCM Round 2)

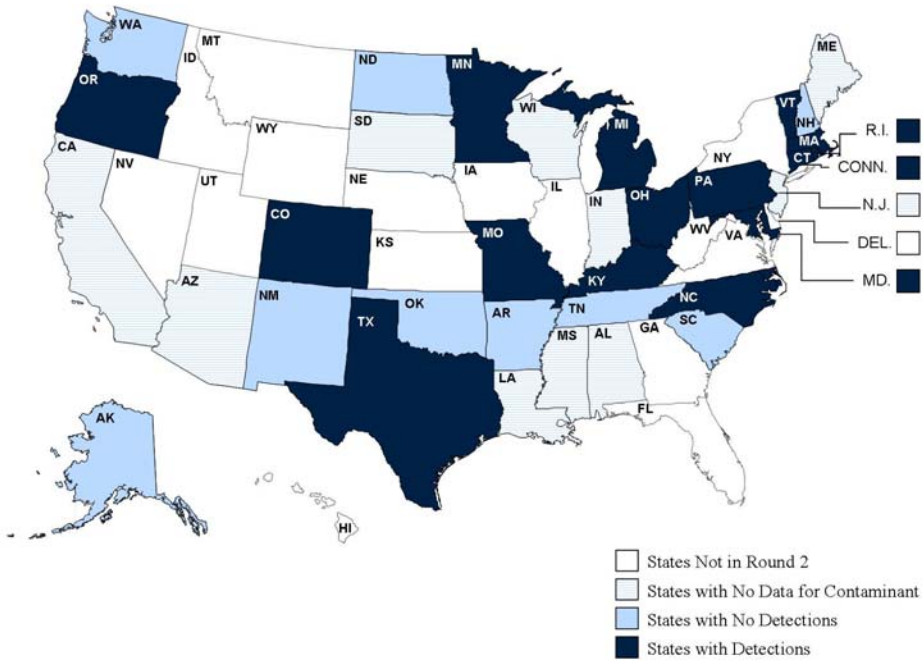
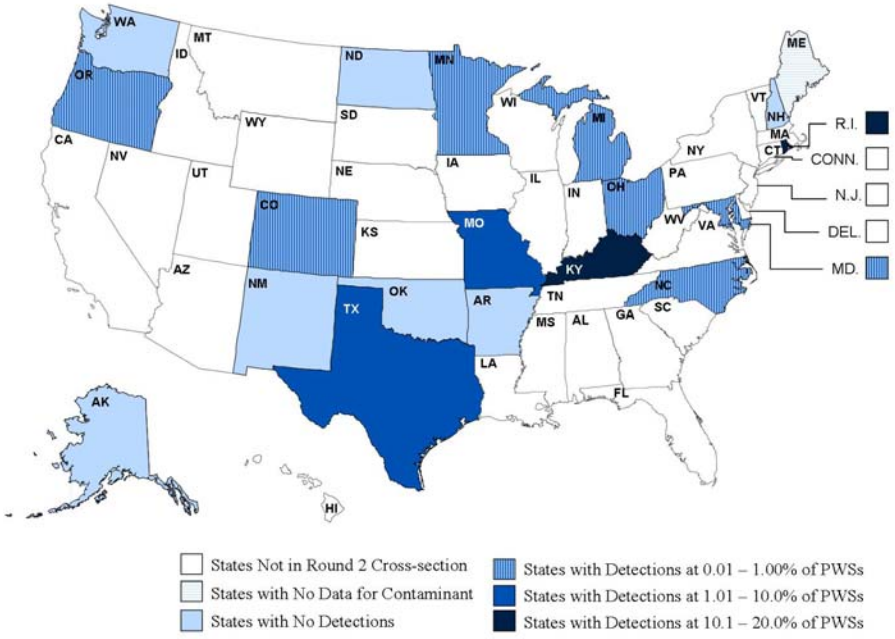
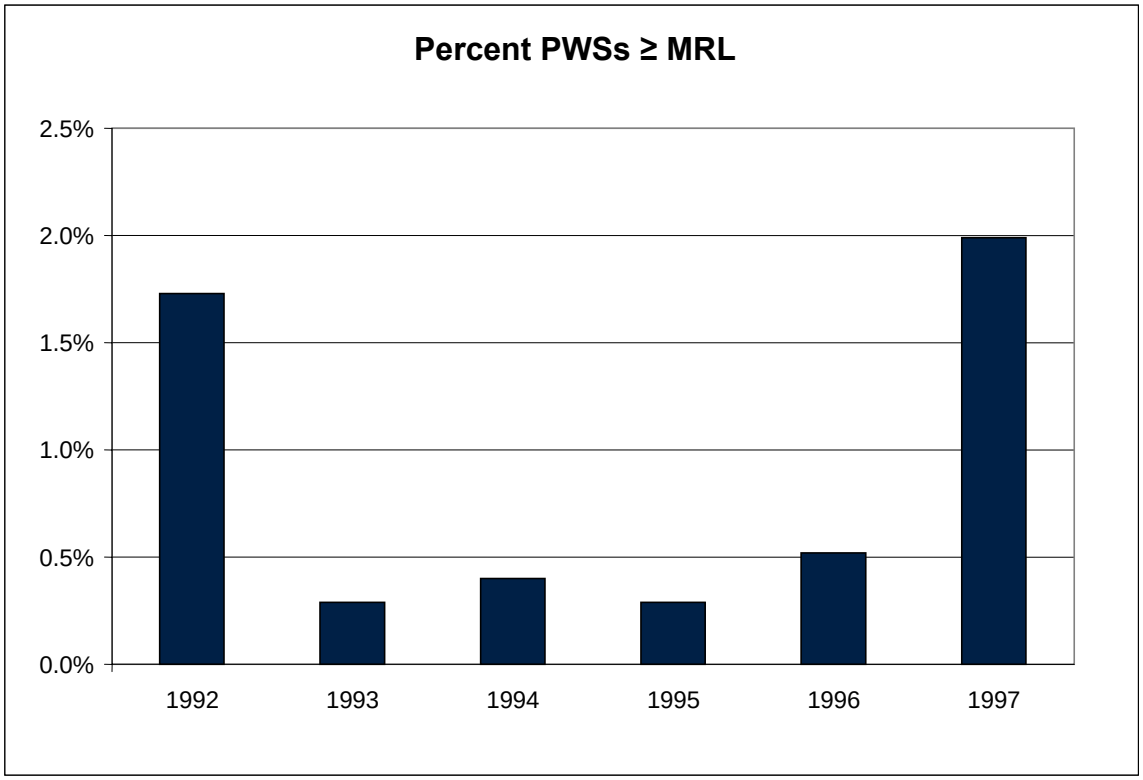


Exhibit 13-8: Geographic Distribution of Metolachlor Detection Frequencies in Cross-Section States (UCM Round 2)



The nineteen States included in Round 2 enable a preliminary temporal assessment of metolachlor occurrence from 1992 to 1997, presented in Exhibit 13-9. The years with the greatest number of PWSs with detections were 1992 and 1997, at the beginning and the end of the monitoring period. A much smaller percentage of PWSs had detections from 1993 through 1996.

Exhibit 13-9: Annual Frequency of Metolachlor Detections, 1992-1997, in Cross-Section States



Pesticides in Ground Water Database (PGWDB)

The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992). Most of the data are from drinking water wells. Since PGWDB data come from multiple sources, they should be interpreted with caution. Results might be biased high, because areas with suspected contamination are likely to have been sampled more frequently than pristine areas. For further information on the PGWDB, see Chapter 2.

According to the data compiled in the PGWDB, metolachlor was detected in 213 (0.96 percent) of 22,255 wells. Metolachlor was found in 20 out of 29 States where monitoring was conducted. The following table shows the range of concentrations by state (USEPA, 1992).

Exhibit 13-10: PGWDB Detections of Metolachlor, 1971-1991

State	No. of Wells with Metolachlor Detections	Range of Detected Concentrations µg/L
Arizona	1	6.9
California	0	-
Connecticut	5	0.2 – 26.0
Delaware	9	0.1 – 12.0
Florida	4	0.150 – 0.520
Georgia	0	-
Iowa	28	0.040 – 22.0
Illinois	7	0.087 – 12.0
Indiana	3	0.3-7.9
Kansas	0	-
Louisiana	0	-
Massachusetts	1	0.24
Minnesota	15	0.10 – 2.4
Maryland	1	120.0
Mississippi	0	-
North Dakota	0	-
Nebraska	6	trace – 2.32
New Jersey	3	0.4 – 1.1
New York	7	0.13 – 112
Ohio	71	0.001 – 6.031
Oklahoma	0	-
Oregon	0	-
Pennsylvania	15	trace – 48
South Dakota	4	0.09 – 0.12
Texas	2	5.3 – 5.7
Virginia	11	0.02 – 2.86
Vermont	6	1.10 – 7.20
Washington	0	-
Wisconsin	14	0.08 – 157.0

National Pesticide Survey (NPS)

EPA collected samples from approximately 1,300 community water system (CWS) wells and rural drinking water wells between 1988 and 1990 for the National Pesticide Survey (NPS). The survey was designed to provide a statistically reliable estimate of pesticide occurrence in the nation's drinking water wells. For details about NPS, see Chapter 2.

With a minimum reporting limit of 0.75 µg/L, metolachlor was not detected in the survey (USEPA, 1990).

Community Water System Survey

The 2000 Community Water System Survey (CWSS) (USEPA, 2002a; 2002b) gathered data on the financial and operating characteristics of a random sample of CWSs nationwide. In addition, the Survey asked all "very large" community water systems, those that serve more than 500,000 people (a total of 83 systems), to provide monitoring results for five regulated compounds (arsenic, atrazine, 2,4-D, simazine, and glyphosate) and four unregulated compounds (radon, methyl tertiary-butyl ether [MTBE], metolachlor, and boron), including results from raw water at each intake and from finished water at each treatment plant. EPA received completed questionnaires from 58 systems. However, not all systems answered every question. Note that because reported results are incomplete, they are more illustrative than statistically representative.

Results of raw water monitoring are aggregated by type of intake. In raw ground water, 4 observations of metolachlor occurrence were reported. Among detects, the median concentration was 1 µg/L and the 90th percentile concentration was 210 µg/L. Non-detects were reported at 44.9 percent of ground water intakes. In raw surface water, 15 observations of metolachlor occurrence were reported. Among detects, the median concentration was 1 µg/L and the 90th percentile concentration was 5 µg/L. Non-detects were reported at 36.7 percent of surface water intakes (USEPA, 2002b).

Results of finished water monitoring are aggregated by system type. At systems primarily served by ground water, 2 observations of metolachlor occurrence were reported. Among detects, the median concentration was 205 µg/L and the 90th percentile concentration was 210 µg/L. Non-detects were reported at 9.1 percent of treatment plants. At systems primarily served by surface water, 20 observations of metolachlor occurrence were reported. Among detects, the median concentration was approximately 0 µg/L (presumably a trace amount) and the 90th percentile concentration was 4 µg/L. Non-detects were reported at 49.5 percent of treatment plants. At systems primarily served by purchased water, there were no reported observations of metolachlor. Non-detects were reported at 67.3 percent of treatment plants (USEPA, 2002b).

Additional Metolachlor Drinking Water Data from the Corn Belt

National metolachlor occurrence data can be augmented by reviewing metolachlor occurrence data collected in the "Corn Belt" States, where metolachlor use is highest. Data from Iowa, Illinois, Indiana, and Ohio are available (Hallberg *et al.,* 1996; USEPA, 1999; Kross *et al.*, 1990; Kolpin *et al*., 1997).

In Iowa, Safe Drinking Water Act (SDWA) compliance monitoring data from surface water and ground water PWSs for the years 1988-1995 reveal that approximately 16 percent of samples analyzed for metolachlor had detections of the compound, with a maximum concentration of 9.4 µg/L. The 99th percentile concentration of all samples was 2.4 µg/L (Hallberg *et al.,* 1996). In a comparison of compliance monitoring data from Illinois, Indiana, and Ohio, mostly collected between 1993 and 1997, the percentage of samples with detections ranged between 0.5 percent for Ohio and 5.2 percent for Illinois. Illinois also had the highest percentage (7.3 percent) of PWSs with detections (USEPA, 1999).

The Iowa State-Wide Rural Well-Water Survey, conducted in 1988-1989 to assess pesticide occurrence in rural private wells, established a statistically significant correlation between increasing well depth and decreasing pesticide contamination, as evidenced by the lower detection frequency of metolachlor in drinking water wells 50 or more feet deep (Kross *et al.*, 1990). This finding is corroborated by the analysis of Illinois compliance monitoring data described above. Although only 7.3 percent of all PWSs in Illinois had metolachlor detections, the rate was approximately 65 percent for surface water PWSs (USEPA, 1999). Nevertheless, data compiled by the Iowa Groundwater Monitoring Program indicate a significant increase in median metolachlor concentration in Iowa ground water from 1982 to 1995. The increase in ground water detections appears to follow the trend of increasing Statewide metolachlor use (Kolpin *et al*., 1997).

13.3.4 Occurrence of Metolachlor Degradates

No national data are available on the occurrence of metolachlor degradates in ambient or drinking water. However, a number of studies have been performed at the local and State level. These can give an indication of the likely occurrence of degradates in areas where metolachlor is used.

In a study by the Wisconsin Department of Agriculture, Trade and Consumer Protection, Wisconsin ground water was sampled from October 1999 to May 2000 for alachlor, acetochlor, metolachlor and their ESA and OA metabolites (Rheineck and Postle, 2000). The 27 monitoring wells, 22 private drinking water wells, and 23 municipal wells sampled for the study were chosen based on past detections of pesticides or proximity to agricultural fields to increase the probability of detecting the pesticides. (These are not, therefore, representative of average occurrence, but are wells of known high occurrence.) Results for metolachlor and its degradates are presented in Exhibit 13-11.

Exhibit 13-11: Wisconsin Ground Water Detections of Metolachlor and Degradates

	Detections	Average Detect (µg/L)	Highest Detect (µg/L)
Metolachlor			
Monitoring Wells	15%	1.7	2.1
Private Drinking Water Wells	36%	1.4	5.9
Municipal Wells	0%	N/A	N/A
Metolachlor ESA			
Monitoring Wells	78%	14	42
Private Drinking Water Wells	91%	4.9	18
Municipal Wells	39%	1.3	4.6
Metolachlor OA			
Monitoring Wells	63%	9.2	32
Private Drinking Water Wells	86%	3.7	23
Municipal Wells	35%	0.57	2.7

Source: Rheineck and Postle, 2000.

In general, the monitoring wells and private drinking water wells showed higher detection frequencies and concentrations than the deeper municipal water wells. Also, the metabolites were detected more frequently and in greater concentrations than the parent compound (Rheineck and Postle, 2000).

A study conducted by Phillips *et al.* (1999a) also found that acetanilide herbicide degradates are detected in higher concentrations than parent compounds. In this study, water samples were collected from April to November 1997 in central New York from tile drains under agriculture fields. Metolachlor ESA was found in a higher range of concentrations than metolachlor OA and the parent compound (3.27-23.4 µg/L versus 1.14-13.5 µg/L and 0.01-0.1 µg/L, respectively).

In 1998, USGS, the New York State Department of Environmental Conservation, and Suffolk County Department of Health Services sampled wells in Suffolk County with known or suspected pesticide residues. Samples were collected from 50 wells that tap the surficial sand-and-gravel water-table aquifer in Suffolk County between May and August. In agricultural areas, at a common reporting level of 0.05 µg/L, metolachlor was detected in more than 35 percent of samples and metolachlor ESA and OA were detected in about 70 percent. In residential and mixed land use areas all three compounds were detected in approximately 10 percent of samples (Phillips *et al.*, 1999b).

13.4 References

Ahrens, W. 1994. *Herbicide Handbook of the Weed Science Society of America*, 7th Edition. Weed Science Society of America, Champaign, IL. (As cited in HSDB, 2004).

Barbash, J.E., G.P. Thelin, D.W. Kolpin, and R. Gilliom. 1999. *Distribution of major herbicides in ground water of the United States*. U.S. Geological Survey Water-Resources Investigations Report 98-4245, 58 p. Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/wrir984245/wrir984245.pdf.

Budavari, S. (ed.). 1996. *The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals*. Whitehouse Station, NJ: Merck & Co., Inc. (As cited in HSDB, 2004).

Chesters, G., G.V. Simsiman, J. Levy, B.J. Alhajjar, R.N. Fathulla, and J.M. Harkin. 1989. Environmental fate of alachlor and metolachlor. *Reviews in Environmental Contamination and Toxicology*. 110:1-74. (As cited in HSDB, 2004).

Eckhardt, D.A.V., W.M. Kappel, W.F. Coon, and P.J. Phillips. 1999. Herbicides and Their Metabolites in Cayuga Lake and its Tributaries, New York State. In: *U.S. Geological Survey Toxic Substances Hydrology Program: Proceedings of the Technical Meeting, Charleston, South Carolina, March 8-12, 1999. Vol. 2 of 3: Contamination of Hydrologic Systems and Related Ecosystems*. Water Resources Investigation Report 99-4018B. Available on the Internet at: http://ny.water.usgs.gov/projects/nypesticides/reports/Eck_WRIR99-4018.pdf.

Extoxnet. 1993. "Extension Toxicology Network Pesticide Information Profiles–Metolachlor." Available on the Internet at: http://pmep.cce.cornell.edu/profiles/extoxnet/metiram-propoxur/metolachlor-ext.html. Accessed December 15, 2004.

Gustafson, D.I. 1989. Groundwater ubiquity score: a simple method for assessing pesticide leachability. *Environmental Toxicology and Chemistry*. 8:339-357. (As cited in HSDB, 2004).

Hallberg, G.R., D.G. Riley, J.R. Kantamneni, P.J. Weyer, and R.D. Kelley. 1996. *Assessment of Iowa Safe Drinking Water Act Monitoring Data: 1988-1995*. Research Report No. 97-1. Iowa City: The University of Iowa Hygienic Laboratory. 132 pp.

Hansch, C., A.J. Leo, and D. Hoekman. 1995. *Exploring QSAR – Hydrophobic, Electronic, and Steric Constants*. Washington, DC: American Chemical Society. (As cited in HSDB, 2004).

Hazardous Substances Data Bank (HSDB). 2004. Search for Metolachlor. Available on the internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine. Available on the Internet at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed November 1, 2004. Last updated May 4, 2001.

Hessler, D.P. and F.H. Frimmel. 1992. American Chemical Society, Division of Environmental Chemistry. Preprint of 203rd ACS National Meeting, 32:121-123. (As cited in HSDB, 2004).

Kalkhoff, S.J., D.W. Kolpin, E.M. Thurman, I. Ferrer, and D. Barceló. 1998. Degradation of chloroacetanilide herbicides - the prevalence of sulfonic and oxanilic acid metabolites in Iowa groundwaters and surfacewaters. *Environmental Science and Technology.* 32(11):1738 -1740.

Kochany, J. and R.J. Maguire. 1994. Sunlight Photodegradation of Metolachlor in Water. *Journal of Agricultural Food Chemistry*. 42:406-412. (As cited in HSDB, 2004).

Kolpin, D.W., D. Sneck-Fahrer, G.R. Hallberg, and R.D. Libra. 1997. Temporal trends of selected agricultural chemicals in Iowa's groundwater, 1982-95: are things getting better? *Journal of Environmental Quality*. 26(4):1007-1017. Available on the Internet at: http://ca.water.usgs.gov/pnsp/ja/jeq26/. Accessed June 18, 2003.

Kolpin, D.W. and J.D. Martin. 2003. "Pesticides in Ground Water: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestgw/Pest-GW_2001_Text.html. Accessed August 24, 2004.

Kross, B.C., G.R. Hallberg, D.R. Bruner, R.D. Libra, K.D. Rex, L.M.B. Weih, M.E. Vermace, L.F. Burmeister, N.H. Hall, K.L. Cherryholmes, J.K. Johnson, M.I. Selim, B.K. Nations, L.S. Seigley, D.J. Quade, A.G. Dudler, K.D. Sesker, M.A. Culp, C.F. Lynch, H.F. Nicholson, and J.P. Hughes. 1990. *The Iowa State-Wide Rural Well-Water Survey Water-Quality Data: Initial Analysis*. Technical Information Series 19. 142 pp.

Lyman, W.J, W.F. Reehl and D.H. Rosenblatt. 1990. *Handbook of Chemical Property Estimation Methods.* Washington, DC: American Chemical Society, pp. 4-9, 15-1 to 15-29. (As cited in HSDB, 2004).

Martin, J.D., C.G. Crawford, and S.J. Larson. 2003. "Pesticides in Streams: Summary Statistics; Preliminary Results from Cycle I of the National Water Quality Assessment Program (NAWQA), 1992-2001." Available on the Internet at: http://ca.water.usgs.gov/pnsp/pestsw/Pest-SW_2001_Text.html. Accessed August 24, 2004.

National Center for Food and Agricultural Policy (NCFAP). 2004. National Pesticide Use Database. Available on the Internet at: http://www.ncfap.org/database/default.htm.

Phillips, P.J., G.R. Wall, E.M. Thurman, D.A. Eckhardt, and J. Vanhoesen. 1999a. Metolachlor and Its Metabolites in Tile Drain and Stream Runoff in the Canajoharie Creek Watershed. *Environment Science and Technology*. 33(20):3531-3735.

Phillips, P.J., D.A. Eckhardt, S.A. Terracciano, and L. Rosenmann. 1999b. *Pesticides and their Metabolites in Wells of Suffolk County, New York, 1998.* U.S. Geological Survey Water-Resources Investigations Report 99-4095, 12 p. Available on the Internet at: http://ny.water.usgs.gov/projects/nypesticides/reports/WRI99-4095.pdf.

Potter, T.L and T.L. Carpenter. 1995. Occurrence of Alachlor Environmental Degradation Products in Groundwater. *Environmental Science and Technology*. 29(6):1557-1563. (As cited in Rheineck and Postle, 2000).

Rheineck, B. and J. Postle. 2000. *Chloroacetanilide Herbicide Metabolites in Wisconsin Groundwater.* Wisconsin Department of Agriculture, Trade, and Consumer Protection. Available on the Internet at: http://datcp.state.wi.us/arm/agriculture/land-water/water-quality/pdf/metabrpt.pdf.

Speth, T.F. and R.J. Miltner. 1990. Adsorption capacity of GAC for synthetic organics. *Journal of American Water Works Association.* 82(2):72-75. (As cited in Speth *et al.*, 2001).

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

SRI International. 2000. *2000 Directory of Chemical Producers*. Menlo Park, CA: SRI Consulting. (As cited in HSDB, 2004).

Syngenta. 2000. Material Safety Data Sheet (MSDS) for Pennant Magnum. Available on the Internet at: http://www.syngentaprofessionalproducts.com/PDF/MSDS/03_12598.pdf.

Thelin, G.P. and L.P. Gianessi. 2000. *Method for Estimating Pesticide Use for County Areas of the Conterminous United States.* U.S. Geological Survey Open-File Report 00-250, 62 p. Available on the Internet at: http://ca.water.usgs.gov/pnsp/rep/ofr00250/ofr00250.pdf.

Thurman, E.M., D.A. Goolsby, D.S. Aga, M.L. Pomes, and M.T. Meyer. 1996. Occurrence of Alachlor and Its Sulfonated Metabolite in Rivers and Reservoirs of the Midwestern United States: The Importance of Sulfonation in the Transport of Chloroacetanilide Herbicides. *Environmental Science and Technology*. 30(2):569-574. (As cited in Rheineck and Postle, 2000).

Tomlin, C.D.S. 1997. *The Pesticide Manual - World Compendium*, 11th Edition. British Crop Protection Council, Surrey, England. (As cited in HSDB, 2004).

Trent, M. and Paulsen, R. 2002. *The Occurrence of Pesticides in Suffolk County*. Water Quality Monitoring Program, Suffolk County Department of Health Services, Suffolk County, New York. Available on the Internet at: http://pbisotopes.ess.sunysb.edu/lig/Conferences/abstracts_02/paulsen.htm. Accessed April 8, 2003.

USEPA. 1990. *National Pesticide Survey: Survey Analytes.* EPA Report 570-9-90-NPS2. Available on the Internet at: http://nepis.epa.gov/pubtitleOSWER.htm. [Search for document number 570990NPS2.]

USEPA. 1992. *Pesticides in Ground Water Database: A Compilation of Monitoring Studies, 1971-1991. National Summary.* Office of Prevention, Pesticides and Toxic Substances. EPA Report 734-12-92-001. Available on the Internet at: http://nepis.epa.gov/pubtitleOPPTS.htm. [Search for document number 7341292001.]

USEPA. 1995. Reregistration Eligibility Decision (RED)--Metolachlor. EPA Report 738-R-95-006. Washington, DC: USEPA Office of Prevention, Pesticides and Toxic Substances. April 1995. Available on the Internet at: http://www.epa.gov/oppsrrd1/REDs/0001.pdf.

USEPA. 1999. *A Review of Contaminant Occurrence in Public Water Systems*. EPA Report 816-R-99-006. 78 pp.

USEPA. 2000. U.S. Environmental Protection Agency/Office of Pesticide Program's Chemical Ingredients Database on Metolachlor (51218-45-2). (As cited in HSDB, 2004).

USEPA. 2001. *The Incorporation of Water Treatment Effects on Pesticide Removal and Transformations in Food Quality Protection Act (FQPA) Drinking Water Assessments*. Available on the Internet at: http://www.epa.gov/oppfead1/trac/science/water_treatment_bak.pdf.

USEPA. 2002a. *Community Water System Survey 2000. Volume I: Overview*. EPA Report 815-R-02-005A. December 2002. Available on the Internet at: http://www.epa.gov/safewater/consumer/pdf/cwss_2000_volume_i.pdf.

USEPA. 2002b. *Community Water System Survey 2000. Volume II: Detailed Tables and Survey Methodology*. EPA Report 815-R-02-005B. Available on the Internet at: http://www.epa.gov/safewater/consumer/pdf/cwss_2000_volume_ii.pdf.

United States Geological Survey (USGS). 2004. 1997 Pesticide Use Maps. Available on the Internet at: http://ca.water.usgs.gov/pnsp/pesticide_use_maps_1997/. Accessed June 3, 2004.

Wauchope, R.D., T.M. Buttler, A.G. Hornsby, P.W.M. Augustijn-Beckers, and J.P. Burt. 1992. The SCS/ARS/CES Pesticide Properties Database for Environmental Decision-Making. *Reviews of Environment Contamination and Toxicology.* 123:1-157. (As cited in HSDB, 2004).

Zimdahl, R.L. and S.K. Clark. 1982. Degradation of three acetanilide herbicides in soil. *Weed Science*. 30:545-548. (As cited in Rheineck and Postle, 2000).

Chapter 14:
MTBE

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Contents

Exhibits

Abbreviations

ADHS	Arizona Department of Health Services
AST	Aboveground Storage Tank
ATSDR	Agency for Toxic Substances and Disease Registry
AWS	American Water System
BTEX	Benzene, Toluene, Ethylbenzene and Xylene
BUST	Bureau of Underground Storage Tanks
CAA	Clean Air Act
California DHS	California Department of Health Services
CAS	Chemical Abstracts Service
CBG	Clean-Burning Gasoline
CCL 2	Second Contaminant Candidate List
Connecticut DPH	Connecticut Department of Public Health
CWS	Community Water System
CWSS	Community Water System Survey
DIPE	Di-isopropyl ether
ETBE	Ethyl tertiary-butyl ether
EWG	Environmental Working Group
Florida DEP	Florida Environmental Protection
GIS	Geographic Information System
HRL	Health Reference Level
IDNR	Iowa Department of Natural Resources
KDHE	Kansas Department of Health and Environment
LARWQCB	Los Angeles Regional Water Quality Control Board
LUST	Leaking Underground Storage Tank
Maine DEP	Maine Department of Environmental Protection
MCL	Maximum Contaminant Level
MDE	Maryland Department of the Environment
MDEQ	Michigan Department of Environmental Quality
MDNR	Missouri Department of Natural Resources
MRL	Minimum Reporting Level
MTBE	Methyl tertiary-butyl ether
NAWQA	National Water Quality Assessment
NDEP	Nevada Department of Environmental Protection
NEIWPCC	New England Interstate Water Pollution Control Commission
NESCAUM	Northeast States for Coordinated Air Use Management
New Jersey DEP	New Jersey Department of Environmental Protection
NHDES	New Hampshire Department of Environmental Services
NPDES	National Pollutant Discharge Elimination System
NTNCWS	Non-Transient Non-Community Water System
PHG	Public Health Goal

PWS	Public Water System
RFG	Reformulated Gasoline
RL	Reporting Limit
SCWC	Southern California Water Company
SRA	Sabine River Authority
STPUD	South Tahoe Public Utility District
TAME	Tertiary-amyl methyl ether
TBA	Tertiary-butyl alcohol
TRI	Toxics Release Inventory
UCMR 1	First Unregulated Contaminant Monitoring Regulation
USDOE	United States Department of Energy
USGS	United States Geological Survey
UST	Underground Storage Tank
VOC	Volatile Organic Compound
Washington DOE	Washington Department of Ecology

14 MTBE

14.1 Definition

Methyl *tertiary*-butyl ether (MTBE) is a volatile organic compound (VOC) commonly used as a gasoline additive. MTBE is also known as methyl *t*-butyl ether, methyl tert butyl ether, and 2-methoxy-2-methylpropane. The Chemical Abstracts Service (CAS) registry number for MTBE is 1634-04-4. It does not have any common trade names.

14.1.1 Properties and Sources

MTBE is a colorless, flammable liquid with a strong, unpalatable odor similar to turpentine. It does not occur naturally in the environment. MTBE is synthesized from methanol, a compound derived from natural gas, and isobutylene or other petroleum refinery products (ATSDR, 1996). Chemically, it is very similar to other ethers such as ethyl *tert*-butyl ether (ETBE) and *tert*-amyl methyl ether (TAME) (USEPA, 2003a). However, because of its low production cost and good blending characteristics, MTBE is the most commonly used oxygenate added to gasoline to improve air quality (Squillace *et al.*, 1997). Like benzene, toluene, ethylbenzene, and xylenes (BTEX), MTBE is also used to increase octane in gasoline (Deeb *et al.*, 2000). Exhibit 14-1 lists some of MTBE's physical and chemical properties and provides a comparison to some of benzene's characteristics.

Exhibit 14-1: Physical and Chemical Properties of MTBE (and Comparison with Benzene)

Identification		
CAS number	1634-04-4	
Molecular Formula	CH_3-O-C(CH_3)$_3$	
Physical and Chemical Properties		
Boiling Point	55.2 °C [1]	
Melting Point	-109 °C [1]	
Molecular Weight	88.15 g/mol [1]	**Benzene**
Log K_{oc}	1.05 (estimated) [2] 2.89 (calculated) [3] 1.04-1.09[4]	1.8-1.99 [4] 1.5-2.16 [4]
Density	0.7405 g/cm^3 at 20 °C [1]	0.8787 g/cm^3 at 15 °C [10]
Log K_{ow}	1.24 [2] 1.20 [4]	2.13 [4] 1.56-2.15 [4]
Water Solubility	51,000 mg/L at 25 °C [5] 43,000 - 54,3000 mg/L [4]	1,780 mg/L [4]
Vapor Pressure	245 mm Hg at 25 °C [6] 245 - 256 mm Hg at 20 °C [4]	76 mm Hg at 25 °C [4] 95.19 mm Hg at 25 °C [4]
Henry's Law Constant	5.87 x 10^{-4} atm-m^3/mol at 25 °C [7] 0.018 (dimensionless) at 20 °C [4] 0.024 (dimensionless), predicted [8] 0.055 (dimensionless), from literature [8]	5.5 x 10^{-3} atm-m^3/mol at 25 °C [11] 0.22 (dimensionless) [4]
Freundlich Isotherm Constant (K)	218 (µg/g)(L/µg)$^{1/n}$ [9]	

[1] *Lide, 1994 (as cited in ATSDR, 1996)*

[2] *Gilbert and Calabrese, 1992 (as cited in ATSDR, 1996)*

[3] *USEPA, 1995a (as cited in ATSDR, 1996)*

[4] *Zogorski et al., 1997*

[5] *Bennett and Philip, 1928 (as cited in HSDB, 2004)*

[6] *Merck, 1989 (as cited in ATSDR, 1996)*

[7] *Hine and Mookerjee, 1975 (as cited in ATSDR, 1996)*

[8] *Speth et al., 2001*

[9] *Speth and Miltner, 1990 (as cited in Speth et al., 2001)*

[10] *Merck, 1989 (as cited in ATSDR, 1997)*

[11] *Mackay and Leinonen, 1975 (as cited in ATSDR, 1997)*

14.1.2 Environmental Fate and Behavior

MTBE has several properties that increase its persistence and mobility in the environment once released. Its Henry's Law constant and high vapor pressure predict volatilization from moist and dry soil surfaces (HSDB, 2004). However, its high water solubility and low Henry's Law dimensionless constant means it dissolves in water more readily rather than it volatilizes (Fiorenza and Rifai, 2003). MTBE is very mobile in soil, only sorbing weakly to soil particles. Benzene's K_{oc} is almost an order of magnitude higher than MTBE's, which indicates that MTBE adsorbs to soil less than benzene.

MTBE primarily reaches ground water via infiltration of gasoline into water, although contamination also occurs via vapor-phase diffusion (Dakhel *et al.*, 2003). Once dissolved in ground water, it generally resists degradation and moves at nearly the same velocity as ground water. Because of its properties, with enough time and distance MTBE would be expected at the leading edge of a gasoline plume, or completely separated from the rest of the plume if the contaminant source were eliminated (Happel *et al.*, 1998).

In 2003 the New England Interstate Water Pollution Control Commission (NEIWPCC) surveyed the 50 States about MTBE contamination at leaking underground storage tank (LUST) sites. Twelve States estimate average MTBE plume lengths of up to 250 feet; 10 States estimate average plumes of 250 to 500 feet, and 2 States (Maine and New York) estimate average MTBE plumes greater than 500 feet. Twenty-six States estimate maximum MTBE-contaminated plumes between 1,000 and 5,000 feet, while 16 States do not know the maximum MTBE plume size (NEIWPCC, 2003).

Other compounds routinely tracked in gasoline spills (e.g., BTEX) are less water-soluble, more volatile (Zanardini *et al.*, 2002), and slightly more retarded by absorption onto soil solids (Squillace *et al.*, 1997). For instance, while the solubility of MTBE in water is about 50,000 mg/L, that of benzene is less than 2,000 mg/L (see Exhibit 14-1). BTEX plumes readily biodegrade, with half-lives ranging from 1 week to 2 years (Howard *et al.*, 1991 as cited in Squillace *et al.*, 1997). Frequently, BTEX plumes stabilize and recede less than 260 feet from the release source (Mace *et al.*, 1997 and Rice *et al.*, 1995, both as cited in Stocking *et al.*, 2000).

Older studies found that MTBE resists degradation under anaerobic methanogenic and sulfate-reducing conditions and anaerobic conditions in landfill-affected aquifer material, soils, and sludges (Squillace *et al.*, 1996). However, MTBE can be degraded in anaerobic conditions by naturally occurring microorganisms if nitrate (Bradley *et al.*, 2001a) or Fe(III) and humic substances (Finneran and Lovley, 2001) are available in the environment. The few anaerobic degradation studies report long incubation times (Zanardini *et al.*, 2002). The British Environmental Agency (2002 as cited in Chisala *et al.*, 2004) concludes from a literature search that anaerobic degradation rates range from 0.0035/day - 0.00035/day (half-lives of 0.54 years to 5.4 years) at hydrocarbon-contaminated sites. The rate of degradation can vary significantly from site to site, and in some cases no biodegradation at all is observed (British Environmental Agency, 2002 as cited in Chisala *et al.*, 2004).

Aerobic biodegradation can occur in the laboratory with MTBE supplied as the sole carbon and energy source (Mo *et al.*, 1997 and Bruns *et al.*, 2001, both as cited in Zanardini *et*

al., 2002), with a low microbial growth rate (Steffan *et al.*, 1997, as cited in Zanardini *et al.*, 2002). Other studies demonstrate co-metabolism of MTBE (Garnier *et al.*, 1999 as cited in Stocking *et al.*, 2000; Hardison *et al.*, 1997; Steffan *et al.*, 1997 as cited in Zanardini *et al.*, 2002). One column study noted that MTBE did not degrade in the presence of BTEX compounds (Church *et al.*, 1999, as cited by Deeb *et al.*, 2000). However, a field study noted that MTBE degraded simultaneously with benzene and toluene when dissolved oxygen increased from 2 mg/L or less up to 6-14 mg/L (Landmeyer *et al.*, 2001). MTBE can biodegrade to carbon dioxide, with intermediate products of tert-butyl alcohol and formaldehyde. Salanitro *et al.* (1998, as cited in Zanardini *et al.*, 2002) note that the toxicity of formaldehyde might limit microbial growth rate. In sediments, the amount of MTBE degradation increased with grain size, perhaps because oxygen diffusion is higher among larger grains (Bradley *et al.*, 2001b). Increasing dissolved oxygen stimulates MTBE degradation in field studies (Salanitro *et al.*, 1999 and Javanmardian and Glasser, 1997, both as cited in Deeb *et al.*, 2000; Wilson *et al.*, 2002).

Ground water containing MTBE commonly has low dissolved oxygen because preferential biodegradation of other fuel components consumes oxygen. Slow field degradation of MTBE may reflect the rate of re-aeration of the groundwater (Moyer and Kostecki, 2003, as cited in Chisala *et al.*, 2004). In field experiments, degradation rates of 0 - 0.007 per day (no degradation to half-lives as short as 99 days) have been reported (Borden *et al.*, 1997, Schirmer *et al.*, 1999, Moeri *et al.*, 2001, all as cited in Chisala *et al.*, 2004). In the laboratory, Schirmer *et al.* (2003, as cited in Chisala *et al.*, 2004) report aerobic rate constants that range from 0.07 to 0.001 per day (equivalent to half-lives of 10 to 693 days). Stocking *et al.* (2000) report aerobic biodegradation half-lives of less than a day to 29 days in the laboratory and 1.6 to 1.9 years in field studies. Several reports also discuss enhanced biodegradation (i.e., Stocking *et al.*, 2000; USEPA, 2001a; Fiorenza and Rifai, 2003; Fayolle *et al.*, 2001).

According to the Blue Ribbon Panel on Oxygenates in Gasoline (1999), limited data suggest that the half-life of MTBE is an order of magnitude longer than the half-life of benzene. Based on this estimate, Johnson *et al.* (2000) conclude that the half-life for MTBE from leaking underground fuel tanks would be at least 2 years. On the other hand, Borden *et al.* (1997) note that at a Sampson County, North Carolina underground storage tank site MTBE degraded from 10 ± 4.6 mg/L at the source to 0.3 ± 0.4 mg/L at points 88 meters downgradient, with little or no further degradation at points beyond. Laboratory experiments using soil from the same site confirmed that under aerobic conditions MTBE degraded from 2.1 mg/L to between 1.0 and 1.5 mg/L within 93 days and then remained relatively constant.

MTBE volatilizes from surface waters. Half-lives in rivers and streams can be greater than one day (Squillace *et al.*, 1996). Factors that affect the volatilization rate of MTBE in surface water include water velocity, water depth, water temperature, wind speed, and air temperature. In deep, slow-moving flows, MTBE volatilizes at rates similar to those of the BTEX compounds. In shallow, fast-moving flows, MTBE volatilizes more slowly than benzene (Squillace *et al.*, 1996). The USEPA's 1998 Research Needs document (USEPA, 1998a) notes that progress is being made on modeling the fate of MTBE in soils, ground water, and surface water.

If released to air, vapor-phase MTBE will be degraded in the atmosphere by reaction with nitrate radicals and photochemically-produced hydroxyl radicals; half-lives for these

reactions in air are estimated to be 50 and 5.5 days, respectively. Direct photolysis is not expected to be an important removal process (HSDB, 2004). According to Squillace *et al.* (1997), the half-life of atmospheric MTBE can be as short as 3 days. Some vapor-phase MTBE will partition to atmospheric water, including precipitation. Squillace *et al.* (1997) observe that atmospheric MTBE can contribute as much as 3 µg/L MTBE to urban precipitation, but that precipitation does not significantly reduce the concentration of MTBE in air.

14.2 Health Effects

In 1997, EPA issued a drinking water advisory of 20 to 40 ppb based on taste and odor (USEPA, 1997). EPA is currently revising its health risk assessment for MTBE. The status of the MTBE health risk assessment can be found on the IRIS Chemical Assessment Tracking System at the following website: http://cfpub.epa.gov/iristrac/index.cfm (USEPA, 2006a).

14.3 Occurrence and Exposure

14.3.1 Use and Environmental Release

MTBE Use: Production and Consumption

MTBE is primarily used as an additive in gasoline to enhance gasoline octane and/or to increase the oxygen content of gasoline . Limited amounts are used medically to dissolve gallstones and as a laboratory solvent for designated EPA analytical methods (ATSDR, 1996). The Agency for Toxic Substances and Disease Registry reports a dramatic increase in MTBE production during the 1980s (ATSDR, 1996). According to the U.S. Department of Energy (USDOE, 2000), MTBE demand increased from 83,000 barrels per day in 1990 to 161,000 barrels per day in 1994 and 269,000 or more barrels per day in 1997. (One barrel is equivalent to 42 U.S. gallons.) Since 1993, MTBE has been the second most widely manufactured organic chemical in the United States (USEPA, 1998b). According to available estimates, annual MTBE production peaked in 1999 (see Exhibit 14-2). Figures compiled by USDOE (2003) indicate that domestic consumption of MTBE also peaked in 1999.

Exhibit 14-2: MTBE Production in the United States

Year	Production in Barrels[1]	Production in Liters
1980	1.65 million barrels[2]	0.26 billion L[5]
1995	68.0 million barrels[5]	10.8 billion L[3]
1998	75.0 million barrels[2,4]	11.9 billion L[5]
1999	78.9 million barrels[4]	12.5 billion L[5]
2000	77.5 million barrels[4]	12.3 billion L[5]
2001	77.5 million barrels[4]	12.3 billion L[5]
2002	74.6 million barrels[4]	11.9 billion L[5]
2003	61.2 million barrels[4]	9.7 billion L[5]
2004	48.1 million barrels[4]	7.6 billion L[5]

[1] One barrel = 42 U.S. gallons

[2] Grady and Casey, 2001

[3] Zogorski *et al.*, 1997 (converted from 8 billion kg using a ratio of 0.74 kg per liter)

[4] USDOE, 2005

[5] Calculated (1 barrel = approximately 159 liters)

Use of MTBE as an octane-enhancing replacement for lead additives in gasoline began in 1979 (USEPA, 1998b). The first standards for a gradual phase-out of lead were issued by EPA in 1973, and the lead ban was complete in 1995 (USEPA, 2005a). Today, regular unleaded gasoline generally contains approximately 3 to 8 percent MTBE (Maine, 1998). MTBE has been used to increase octane throughout the U.S. (Landmeyer *et al.*, 2001). Other octane enhancers widely used in gasoline include ethanol, alkylates, and aromatic compounds. According to one U.S. Department of Energy report, in 1997 approximately 12,000 barrels of MTBE per day, or close to 5 percent of total MTBE consumption, went toward octane enhancement in conventional gasoline (Lidderdale and Bohn, 1999; 65 FR 16097). A subsequent report suggests that the amount of MTBE used to raise octane in conventional gasoline has varied significantly from year to year, rising from negligible amounts in 1995 to approximately 46,500 gallons, or 16 percent of total MTBE consumption, in 2001 (Lidderdale, 2003).

In the 1990s, MTBE use increased due to two programs established by the 1990 Amendments to the Clean Air Act (CAA) that require oxygenated gasoline. In 1992 EPA implemented the Wintertime Oxygenated Fuel (Wintertime Oxyfuel) program for metropolitan areas with elevated levels of carbon monoxide. About 4 percent of the nation's gasoline is oxyfuel. The oxyfuel program requires gasoline to have an oxygen content of 2.7 percent by weight (USEPA, 1998b). Although ethanol is the most common oxygenate used to meet this requirement (7.3 percent by volume), MTBE is also sometimes used at concentrations of up to 15 percent by volume (USEPA, 1998b; 65 FR 16097).

In 1995 EPA established the Federal Reformulated Gasoline (RFG) program, which requires gasoline used in the nation's most polluted metropolitan areas to contain 2 percent oxygen by weight. In 1998 this requirement applied to about 30 percent of the nation's gasoline. The requirement can be met with 11 percent MTBE or 5.4 percent ethanol (by volume) (USEPA, 1998b). MTBE is the primary oxygenate in over 87 percent of RFG, while ethanol is used in approximately 12 percent (65 FR 16097). The "non-attainment areas" required to participate in the program are metropolitan areas where ozone levels are too high. Other areas of the country voluntarily "opt in" to the RFG program to improve air quality. The total number of areas participating in the RFG program may change from year to year, depending on "opt-ins" (USEPA, 2000a). A list of participating and formerly participating areas is available on the Internet (USEPA, 2005b). There is considerable variation in the extent to which participating areas rely on MTBE. Some areas are in States or localities that have implemented MTBE bans (so other oxygenates are used). In Chicago, for example, MTBE was banned in 2000, and four years later a Statewide ban went into effect in Illinois (Lidderdale, 2003).

Information on the geographic distribution of MTBE consumption (see Exhibit 14-3) is available from Lidderdale (2003). In 2001, approximately 33 percent of the MTBE used to comply with Clean Air Act requirements was consumed in California. At that time, California had State emission standards that were stricter than the federal program, but had not yet banned MTBE. Other States consuming large amounts of MTBE in RFG and Oxygenated Fuel in 2001 included Texas (13 percent of U.S. consumption), New Jersey (11 percent), New York (9 percent), and Massachusetts (7 percent). The twelve Northeastern and Mid-Atlantic States studied in depth by the United States Geographical Survey (USGS) (Grady and Casey, 2001--see study description in Section 14.3.3, below) accounted for about 50 percent of the MTBE used to comply with Clean Air Act requirements in 2001.

Exhibit 14-3: Consumption of MTBE in RFG and Oxygenated Fuel in 2001

State	MTBE use (in 1,000 barrels/day)
Arizona	3.6
California	79.7
Connecticut	9.4
Delaware	3.0
District of Columbia	0.7
Kentucky	2.2
Maryland	12.6
Massachusetts	16.8
Montana	3.2
New Hampshire	3.2
New Jersey	27.1
New York	21.1
Pennsylvania	9.7
Rhode Island	2.6
Texas	30.5
Virginia	13.6

Note: These numbers do not include MTBE used for octane enhancement in conventional gasoline. MTBE is one of several oxygenates used to meet federal RFG and Oxygenated Fuel requirements.

Source: Lidderdale, 2003

The Energy Policy Act of 2005 (also known as the "Energy Bill," or H.R.6), signed on August 8, 2005, amended the CAA. Some of the amendments are expected to reduce the amount of MTBE used in gasoline. In particular, Section 211(k)(2) of the CAA no longer contains minimum oxygen content requirements for reformulated gasoline (RFG) (see 71 FR 8965 and 71 FR 26691). However, RFG must maintain the pollutant emissions reductions required by Section 211(k)(1). Unlike earlier House drafts, the bill does not give "safe harbor" to protect manufacturers of gasoline containing MTBE from defective product liability suits filed because of drinking water contamination (McCarthy and Tiemann, 2005). In response, at least one major U.S. oil refiner has announced it would cease MTBE production altogether (Vaughan, 2005). In addition, at least 25 states have passed laws banning or limiting the use of MTBE, with effective dates ranging from 2000 to 2009 (see Exhibit 14-22, below). The Department of Energy projects that MTBE use in gasoline will be entirely phased out in the United States by the end of 2008 (USDOE, 2006).

MTBE Releases to the Environment

MTBE's widespread use as a gasoline additive provides a number of opportunities for release of MTBE to the nation's ground and surface waters. According to the Alliance for Proper Gasoline Handling (1999), each year approximately 9 million gallons of gasoline are released to the environment in the United States from leaks and spills. Leakage from gasoline storage and distribution systems is a major source of both aboveground and underground contamination.

Underground storage tanks (USTs) and other gasoline storage and distribution facilities are responsible for releasing large volumes of gasoline into the environment. In 1984, the State

of Maine estimated national releases from leaking USTs to be 11 million pounds (Feliciano, 1984 as cited in ATSDR, 1995). More recent estimates are not available. Releases cause high concentrations of MTBE in soil and ground water relatively near the contaminant release (or "source") area, and in the MTBE-containing plumes that extend outward from the source. There are approximately 760,000 regulated gasoline USTs in the U.S. In addition, there are approximately 3 to 4 million underground fuel storage tanks (*e.g.*, smaller farm and residential gasoline storage tanks and home heating oil tanks) exempt from Federal regulations (65 FR 16100). Some States regulate heating fuel tanks. A 2000 survey of State UST program offices by the NEIWPCC, funded by EPA's Office of Underground Storage Tanks, indicates that 35 States find MTBE at least 20 percent of the time they sample for it in ground water at gasoline-contaminated sites, and 24 States find MTBE at least 60 percent of the time, out of 46 States providing responses to that question (NEIWPCC, 2000). Results from a follow-up survey in 2003 are comparable (NEIWPCC, 2003). Forty-one States indicated that they request analysis for MTBE in ground water 80 to 100 percent of the time at LUST sites for at least one type of fuel (gasoline, heating oil, jet fuel, etc.) (NEIWPCC, 2003). In California, 78 percent of LUST sites where gasoline has impacted ground water are positive for MTBE (Happel *et al.*, 1999). See Exhibit 14-4 for a comparison of the number of States reporting contamination (at any concentration) at public and private drinking water wells in 2000 and 2003.

Exhibit 14-4: MTBE Contamination Reported by States

	2000 Survey		2003 Survey	
	private wells	**public wells**	**private wells**	**public wells**
Number of States reporting no detections at wells	3	5	3	3
Number of States reporting detections at between 1 and 50 wells	14	21	12	19
Number of States reporting detections at more than 50 wells	9	6	17	7
State reporting the highest number of contaminated wells	New York (866 wells)	Connecticut (255 wells)	New Hampshire (over 30,000 wells)	New Hampshire (350 wells)
Number of States that "don't know"	16	16	13	13
Total number of responding States	42	48	44	42

Note: In the 2000 survey, Puerto Rico was included among responding "States." In the 2003 survey, Idaho is double-counted, as both "none" and "between 1 and 10 private wells."

Sources: NEIWPCC, 2000 (question 16a); NEIWPCC, 2003 (question 27)

In 2000, twenty-five State UST program offices reported finding at least one case of soil or ground water MTBE contamination even where no release was documented (NEIWPCC, 2000). In 2003, twenty-three offices reported finding contamination that could not be attributable to USTs; suspected sources included above-ground storage tanks (ASTs), auto accidents, auto maintenance, lawn mowers, and improper handling and storage (NEIWPCC, 2003).

EPA requirements for USTs were phased in between December 1988 and December 1998 (USEPA, 1995b). Vapor recovery systems capture vapors released during the filling of the UST (Stage I) or the fueling of vehicles (Stage II) and return them to the vessel the liquid fuel came from (i.e., the tank truck or UST). Stage II vapor recovery systems are required in moderate or worse ozone non-attainment areas (USEPA, 2004a). Some vapor recovery systems, called "assisted" or "vacuum-assist" systems, use vacuum pumps to capture fumes from the vehicle's gasoline tank. EPA estimates that by September 2004, approximately 64 percent of active UST systems were in significant operational compliance with all leak detection and release prevention requirements (USEPA, 2005c). However, according to recent studies, summarized below, even fully compliant USTs can be sources of MTBE contamination. Recent research has focused on gasoline USTs and the role of vapor releases. With a pure-phase vapor pressure of 251 mg Hg, MTBE volatilizes more readily than BTEX compounds and ethanol, whose pure-phase vapor pressures are 2.5 to 31 times lower. The partial pressure of a constituent compound of gasoline is equal to its pure-phase vapor pressure multiplied by its fractional content in the gasoline. When MTBE is used in gasoline, it is often present at concentrations higher than the BTEX compounds, which makes it volatilize even more readily (Day *et al.*, 2001; API, 2000).

Levine-Fricke (1999) conducted a preliminary investigation of in-compliance UST facilities in Santa Clara, California, and found evidence of MTBE ground water contamination at 13 of 27 facilities. MTBE concentrations ranged from 1 µg/L to 200,000 µg/L; five facilities had concentrations above 1,000 µg/L. The study found a statistically significant correlation between facilities with assisted vapor recovery systems and facilities with MTBE contamination. A follow-up study by Tulloch (2000) evaluated 16 active UST facilities in Santa Clara known to have high concentrations of MTBE in ground water to determine the likely source and cause of contamination. The investigators found that in 13 of the 16 cases the contamination was likely due to undetected releases from the current UST system. In two cases the points of release were positively identified. In other cases no point of release could be confirmed, but inspection revealed deficiencies in tanks, sumps, lined trenches, and/or piping that could be responsible. Nine of the USTs had records of monitoring problems or violations. The investigators concluded that both single-walled and double-walled systems can fail, and that increased vigilance is necessary.

In a statewide California study, Young and Golding (2002) used a sensitive commercial leak detection method to monitor vapors at 182 USTs at 55 randomly selected facilities. Releases (as determined by the presence of a tracer chemical) were observed at 61 percent of the USTs and at 80 percent of the facilities. One release was a liquid phase release from a single-walled UST. The remaining cases were vapor-phase releases, and double-walled tanks appeared to be no better at preventing releases than single-walled tanks. The largest release was a vapor release of 0.4 gallons per day. Most releases (including the liquid release) were less than 0.04 gallons per day. It is likely that none of the releases observed in this study would be detected by conventional leak detection methods, since the greatest sensitivity required of leak detection methods under current regulations is 0.1 gallons per hour (or 2.4 gallons per day). Components of the tank top were the most likely source of tracer releases. According to Lynn (2004), Vermont remedial UST investigations confirm that vapor could escape from many tank-top components, including vent lines, ancillary risers, caps, in-tank monitor wiring fittings, and Stage I vapor-recovery poppets.

In an investigation of five USTs, the New Hampshire Department of Environmental Services (NHDES) (Lynn, 2004) determined that both USTs with vacuum-assist vapor recovery systems and those without vacuum-assist recovery systems had positive pressure. Further investigation of two vacuum-assist USTs with vapor releases showed that pressure decreased at night, suggesting that vapors were escaping the tank system. DES confirmed this using a technology to manipulate tank pressure and a tracer chemical. When pressure in the tank system was reduced, the amount of vapors released into the soil diminished.

Transport provides additional opportunities for potential MTBE contamination. Pipelines are used to transport billions of gallons of crude oil and refined products annually in the United States. Gasoline may travel through thousands of miles of pipelines, or be transported by truck to terminals and bulk stations. From there, it may be transported to fleet storage facilities, retail outlets, or above- or underground tank facilities by truck. Spills and accidental releases can occur at any point in these processes. Residual gasoline in transport conduits may also contaminate different types of fuels that are transported through the same conduits at other times, which may explain the presence of MTBE in home heating oil, jet fuel, and diesel fuel (65 FR 16100). The Michigan Department of Environmental Quality (MDEQ) surmised that gasoline containing less than 2.2 percent MTBE by volume results from mixing in pipeline distribution (MDEQ, 2000 as cited in Delzer and Ivahnenko, 2003a).

Releases from automobile accidents, tank truck spills, improper consumer disposal, and spills during fueling operations have been identified as sources of contamination of drinking water wells. Gasoline-powered watercrafts contribute to MTBE contamination of lakes and reservoirs. For example, Shasta Lake, California, reached 9-88 µg/L MTBE over the Labor Day weekend in 1996. Pre-1998 outboard and personal watercraft two-stroke engines can discharge up to 30 percent of gasoline as unburned hydrocarbons. Stormwater runoff and air deposition can also play a role in low-level MTBE contamination of water resources (65 FR 16096). Zogorski *et al.* (1997) report that ambient air concentrations of MTBE in a number of U.S. cities range from 0.13 to 4.6 parts per billion by volume.

When used as a gasoline additive, MTBE concentrations are high, starting at 20,000,000 parts per billion (ppb) for 2 percent volume to improve octane. Exhibit 14-5 compares some MTBE concentrations at the high end of the ranges found in various media.

Exhibit 14-5: Examples of High MTBE Concentrations in Various Media

Where Found	MTBE concentration	Comments
Oxygenated Gasoline[1]	110,000,000 ppb - 150,000,000 ppb	typically 11% to 15% by volume (API, 2000)
Conventional Gasoline[1]	20,000,000 ppb - 30,000,000 ppb	typically 2% to 3% by volume, when added for octane enhancement (API, 2000)
Ground Water	9,132,000 µg/L	measured at the core of a LUST plume in Texas (NEIWPCC, 2003)
Ground Water	1,000,000 µg/L	plumes with concentrations higher than this are rarely seen higher in the field, according to API (2000)
Ground Water	200,000 µg/L	measured near a gasoline spill (Zogorski *et al.*, 1997)
Ground Water	200,000 µg/L	measured near an in-compliance UST (Levine-Fricke, 1999)
Ground Water	100,000 µg/L	20 States report finding concentrations higher than this at the center of LUST plumes (NEIWPCC, 2003)
Ground Water	64,000 µg/L	measured near an in-compliance UST (Shively, 2004)
Private Well Water	6,500 µg/L	measured near an auto accident (65 FR 16099)

[1] As presented here, MTBE concentrations in gasoline are not converted from parts per billion (ppb) to micrograms per liter (µg/L). In water, 1 ppb = 1 µg/L (because 1 gram is the mass of 1 cubic centimeter of water and 1,000 cubic centimeters = 1 liter), but in gasoline, a less dense fluid, this equivalency does not hold. Nevertheless, if MTBE concentrations in gasoline were converted to µg/L, the results would be on the same order of magnitude as the ppb values presented here.

MTBE is listed as a Toxics Release Inventory (TRI) chemical. For a discussion of the nature and limitations of TRI data, see Chapter 2. TRI data for MTBE (see Exhibit 14-6) are reported for the years 1988 to 2003. On-site air emissions dominate total releases; these peaked in 1998 at over four million pounds. Annual releases by other routes fluctuated between hundreds of pounds and hundreds of thousands of pounds during the period on record. Releases of MTBE were reported from all States except North Dakota and Vermont (USEPA, 2006b).

Exhibit 14-6: Environmental releases (in pounds) of MTBE in the United States, 1988-2003

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1988	2,588,247	21,499	14,400	370	4,602	2,629,118
1989	3,223,014	37,440	19,300	1,290	4,623	3,285,667
1990	2,976,906	42,668	112,400	1,501	7,696	3,141,171
1991	3,270,121	30,903	81,690	2,903	6,348	3,391,965
1992	3,139,291	102,869	68,445	288	15,329	3,326,222
1993	3,780,143	94,261	9,406	409	134,331	4,018,550
1994	3,188,678	92,140	29,645	2,226	117,854	3,430,543
1995	3,300,759	78,555	15,238	3,800	47,841	3,446,193
1996	3,098,099	117,760	179,624	26,569	243,430	3,665,482
1997	2,658,874	162,116	16,720	124	119,851	2,957,685
1998	4,225,523	66,347	51,707	3,209	265,679	4,612,465
1999	3,779,740	121,186	20,677	6,606	261,297	4,189,506
2000	3,462,233	123,536	33,336	10,556	34,756	3,664,417
2001	3,064,142	67,191	49,677	4,255	35,901	3,221,165
2002	2,957,648	68,693	79,089	1,527	80,570	3,187,527
2003	2,432,275	60,617	83,905	52,241	142,223	2,771,261

Source: USEPA, 2006b.

Although MTBE production has been declining since 1999 and the use of MTBE in gasoline is expected to cease by 2008 (see above), contamination of drinking water wells is likely to continue because of existing soil and ground water contamination and continued MTBE-contaminated plume movement. Johnson *et al*. (2000) place the time scale of the threat of contamination in the range of tens to hundreds of years, based on the expected lifetime of LUSTs, the concentration of MTBE in such sources, estimated rates of attenuation in ground water, and the potential distance plumes might travel before reaching community water systems (CWSs).

14.3.2 Ambient Water Occurrence

Ambient waters are lakes, rivers, and aquifers that serve as sources of drinking water. Data on the occurrence of MTBE in ambient surface and ground water are available from the National Water Quality Assessment (NAWQA) program of the USGS. For details on this program, see the discussion in Chapter 2. NAWQA data have been analyzed independently by USGS and EPA. USGS has also collected data on MTBE occurrence in a review of highway and urban runoff studies. For additional perspective, State studies are also summarized in this section.

USGS NAWQA VOC National Synthesis

Random and Focused VOC Surveys and Literature Review

In collaboration with the Metropolitan Water District of Southern California and the Oregon Health & Science University, and with funding from the American Water Works Research Foundation, USGS recently conducted an assessment of the occurrence of MTBE and other VOCs in the nation's source waters. The assessment included a random survey of VOC occurrence in ground and surface water resources used by geographically representative CWSs in different size categories (Grady, 2003) and a focused survey of VOC occurrence patterns, including seasonal variability, in source waters considered particularly susceptible to MTBE contamination (Delzer and Ivahnenko, 2003b). Analytes included MTBE, three other ether oxygenates used in gasoline, and 62 additional VOCs. The reporting limit for MTBE was 0.2 µg/L (Ivahnenko *et al.*, 2001). The effort, which relied on data from NAWQA study units and others sources, was the culmination of USGS recently completed national VOC sampling program (or VOC National Synthesis).

The random survey sampled MTBE occurrence in ground and surface water sources used by 954 geographically representative CWSs in different size categories (Grady, 2003). Following a quality control review, 934 of the 954 samples were used to determine MTBE occurrence. At the reporting limit of 0.2 µg/L (25 times lower than the UCMR 1 minimum reporting level), the random survey of source waters found MTBE in 8.7 % of the samples (5.4% of ground water samples and 14% of surface water samples). Among detections, the median concentrations in ground water, reservoirs, and rivers were 0.71 µg/L, 0.67 µg/L, and 0.32 µg/L, respectively (Grady, 2003). Only 3 samples (0.3%) had concentrations that exceeded 5 µg/L. These were from a surface water site in Texas (6.12 µg/L), a ground water site in Florida (6.31 µg/L), and a surface water site in California (19.5 µg/L) (USGS, 2003). Grady (2003) provides detection statistics for five different size categories. Aggregating these categories, it can be calculated that source waters of systems serving over 10,000 people had a combined detection frequency of 14.4%, while source waters of systems serving 10,000 people or fewer had a combined detection frequency of 4.2%. MTBE detections were five times more frequent in areas with high MTBE use than in areas with low or no MTBE use.

The focused survey investigated 134 CWS source waters (56 surface and 78 ground water) between 1999 and 2001 (Delzer and Ivahnenko, 2003b). Included were 57 source waters known to contain MTBE (including source waters found to have MTBE levels above 0.5 µg/L in the random survey), plus 77 source waters thought likely to be contaminated with MTBE, usually on the basis of high population density and high current or historical rates of MTBE use in gasoline. Each of 78 groundwater sites was sampled twice during a one-year period; 39 reservoir and lake sites were each sampled quarterly for one year, and 17 stream sites were each sampled eight times per year for one year. At the reporting limit of 0.2 µg/L, MTBE detections were found in 55.5% of sites (60.0% of ground water sites and 49.1% of surface water sites) (Delzer and Ivahnenko, 2003b). In ground water, the highest MTBE concentration was either 924 µg/L (as listed in Table D-1 of the published report) or 926 µg/L (according to the focused study data made available to the public in spreadsheet form) (Delzer and Ivahnenko, 2003b; USGS, 2003). Seventeen groundwater systems had MTBE detections that exceeded 5 µg/L, and seven of these systems had detections greater than 20 µg/L. The highest MTBE concentration in

surface water was 14 µg/L. Four surface water systems had detections that exceeded 5 µg/L (USGS, 2003). Although the presence of gasoline oxygenates as a class varied seasonally in lakes and reservoirs, a phenomenon attributed by the study authors to increased use of motorized watercraft during the spring and summer months, no seasonal MTBE occurrence patterns were detected in any type of source water (Delzer and Ivahnenko, 2003b).

Before assessing MTBE in the nation's drinking water supplies, USGS conducted a literature review (Delzer and Ivahnenko, 2003a). Of greatest interest here (since most other studies in the literature review are discussed elsewhere in this chapter) is a compilation of results from individual States. Thirteen States conducted their own assessments of MTBE occurrence, primarily in source water. The State reports ranged from full inventories of CWS drinking water and selected private residential wells to results of contaminated domestic wells and voluntary sampling of source waters (see Exhibit 14-7). The States reported maximum MTBE concentrations of 8.39 µg/L in Alabama, 610 µg/L in California, 110 µg/L in Connecticut, 166 µg/L in Florida, 63 µg/L in Iowa, 1,250 µg/L in Kansas, 30.2 µg/L in New Jersey, and 1,700 µg/L in Wisconsin. Illinois, Maine, Maryland, Michigan, and Missouri also submitted data, but not enough to determine a maximum concentration. USGS noted that study findings from the various states could not be directly compared or used to estimate national exposures, given the different reporting levels, sampling frequencies, and sources (public source water, private wells, etc.).

Exhibit 14-7: Statewide Assessments of MTBE

State Survey Summary	Reporting Limit (RL)	Detection Frequency	Median Detected Concentration	Maximum Detected Concentration
Alabama: complete 2000 survey of 575 PWSs. Sampling at 1,053 sources (87 surface water sources, 27 springs, 939 wells)	0.5 - 2.0 µg/L	wells: 0.53% springs: 0% surface water sources: 0%	wells: NA springs: NA surface water sources: NA	wells: 8.4 µg/L springs: NA surface water sources: NA
California: partial survey of PWS source waters, covering 105 of 245 surface water sources (3,000 samples) and 2,988 of 13,919 PWS wells in 1996-1997; supplemented by mixed surface and ground water info from DHS database (50,748 samples collected between 1989 and 2001)	NA DHS database: NA	surface water sources: 46.7% wells: 1.2% DHS database: 1.1%	surface water sources: NA wells: NA DHS database: 3.6 µg/L	surface water sources: > 14 µg/L (26%) wells: NA DHS database: 610 µg/L
Connecticut: 1999 annual report on organics testing at PWSs (total number of PWSs not reported)	0.5 - 2.0 µg/L	NA (detected in 57 sources waters in 40 towns)	2.7 µg/L	110 µg/L
Florida: 8,739 samples collected from 1,692 public water supplies since early 1990s.	NA	4.9% of samples, 1.2% of PWSs (89% of the detects were from two PWSs)	1.4 µg/L	166 µg/L

State Survey Summary	Reporting Limit (RL)	Detection Frequency	Median Detected Concentration	Maximum Detected Concentration
Illinois: monitoring since 1994 at approximately 80% of the State's 1,200 CWSs, most of which (92%) utilize ground water	0.5 - 1.0 µg/L	2.7% of active systems, plus three systems that abandoned wells following MTBE contamination	NA	NA (IL states that most of the concentrations were unlikely to cause adverse health effects)
Iowa: 530 samples collected from 235 PWS wells in "vulnerable bedrock regions" in 1999; plus sampling of drinking water supplies in several cities since the 1990s	bedrock project: 15 µg/L cities: NA	bedrock project: 8 sample detections < 15 µg/L cities: NA	bedrock project: < 15 µg/L cities: NA	bedrock project: < 15 µg/L cities: 63 µg/L in Alvord's water supply before well abandoned
Kansas: 27,935 samples from 1,122 PWS wells, collected 1996 - 2000	NA	1.6% of wells	NA	1,250 µg/L
Maine: survey of 793 of 830 public water supplies and 951 private household water supplies in 1998	0.1 µg/L	public supplies: 15.8% (6% had concentrations in the range of 1-35 µg/L) private supplies: 15.8%; (6.6% had concentrations in the range of 1-35 µg/L)	public supplies: NA private supplies: NA	public supplies: < 35 µg/L private supplies: > 35 µg/L (1.1% of supplies)
Maryland: 1,084 PWSs surveyed since 1995; data also collected on private wells contaminated by LUSTs	0.5 µg/L	PWSs: 7.8% private wells: NA (270 wells with detections out of an unspecified number sampled)	PWSs: NA private wells: NA	PWSs: > 20 µg/L (11 systems) private wells: NA
Michigan: 31,557 samples from 18,046 CWS, NCWS, and private wells from 1987 through 1999	1.0 µg/L	2.9% of samples and 3.0% of wells	NA	> 240 µg/L (29 samples)
Missouri: MO has monitored MTBE in 1,685 PWSs since 1994	5 µg/L	0.1% of monitored PWSs statewide (2 PWSs)	NA	NA
New Jersey: samples from approximately 400 CWSs from 1997 to 1998; plus a random sampling of 104 domestic wells	PWSs: 0.5 µg/L private wells: 0.1 µg/L	PWSs: 14.8% private wells: 35.6%	PWSs: NA private wells: 0.48 µg/L (average of medians from 4 sampling areas)	PWSs: 8.4 µg/L private wells: 30.2 µg/L
Wisconsin: 2,271 wells (mostly private) sampled since 1990	12 µg/L	4.4% of wells (96 private wells and 3 public wells)	NA	1,700 µg/L (private well)

NA = not available
Source: Delzer and Ivahnenko, 2003a (see this literature review for full citations for State studies)

Compilation of Historical VOC Monitoring Data

USGS assessed VOC occurrence in untreated ambient ground water samples collected between 1985 and 1995 by local, State, and federal agencies (Squillace *et al.*, 1999). The samples represented both urban and rural areas, and both drinking water and non-drinking water wells. MTBE samples were collected from 225 urban wells and 1,312 rural wells. Of the 60 VOCs monitored at a reporting level of 0.2 µg/L, MTBE was one of the most frequently detected VOCs, particularly in urban areas. There was a 16.9 percent detection frequency in urban areas and a 3.4 percent detection frequency in rural areas. Detections ranged from the minimum reporting level (0.2 µg/L) to over 10,000 µg/L. The median detected concentration was less than 1 µg/L.

EPA Summary Analysis of NAWQA Data

While the NAWQA program often uses the most representative data for a site to calculate summary statistics, EPA, with the cooperation of USGS, has performed a summary analysis of all Cycle 1 water monitoring data from all study units (1991-2001) for many of the Second Contaminant Candidate List (CCL 2) contaminants being considered for regulatory determination, including MTBE. Detection frequencies were simply computed as the percentage of samples and sites with detections (i.e., the percentage with at least one result equal to or greater than the reporting limit; note that reporting limits were not uniform). Sample detections can be biased by frequent sampling in areas with high (or low) occurrence. Calculating the percentage of sites with detections can reduce this bias. For more details on the NAWQA data set and the EPA analysis, see Chapter 2.

The results of the EPA analysis are presented Exhibit 14-8. Overall, MTBE was detected in 17.7 percent of samples and at 9.8 percent of sites. MTBE was detected more frequently in surface water but at higher concentrations in ground water.

Exhibit 14-8: EPA Summary Analysis of MTBE Data from NAWQA Study Units, 1992-2001

	Detection Frequency (detections are results ≥ RL[1])				Concentration Values (of detections, in µg/L)				
	Number of Samples	% Samples with Detections	Number of Sites	% Sites with Detections	Minimum	Median	95th Percen-tile	99th Percen-tile	Maximum
surface water	1,402	46.2%	182	62.1%	0.01	0.25	3.46	63	81.3
ground water	4,645	9.1%	4,146	7.5%	0.01	0.5	800	4,500	23,000
all sites	6,047	17.7%	4,328	9.8%	0.01	0.3	320	1,800	23,000

[1] RLs (Reporting Limits) for MTBE varied, but did not exceed 0.2 µg/L. For more information, see Chapter 2. Note that because this EPA analysis involves more data points than the USGS analyses presented above, a direct comparison is not possible. Some concentration values are rounded.

USGS Stormwater Studies

For the National Highway Runoff Data and Methodology Synthesis, USGS conducted a review of 44 highway and urban runoff studies implemented since 1970 (Lopes and Dionne, 1998). Three of these studies report results for MTBE.

Two of the three studies with MTBE results were stormwater studies conducted in specific major metropolitan areas in connection with National Pollutant Discharge Elimination System (NPDES) permitting. In metropolitan Phoenix (Maricopa County), USGS collected 35 samples from five drainage basins, and the City of Phoenix collected an additional 26 samples from seven sites (Lopes *et al.*, 1995). In Dallas-Fort Worth, 182 samples were collected from 26 stormwater drainage basins (Baldys *et al.*, 1998). The reporting limits were 0.2 µg/L in Phoenix and ranged from 0.2 to 10 µg/L in Dallas-Fort Worth. Not all samples were monitored for every contaminant. The maximum detected MTBE concentrations were 2.5 µg/L in Phoenix and 8.7 µg/L in Dallas-Fort Worth.

The third study was a summary of all NPDES-related stormwater monitoring data on MTBE and other compounds from sixteen cities from 1991-1995, including the two described above (Delzer *et al.*, 1996). This summary was undertaken to support an interagency assessment of the scientific basis and effectiveness of the nation's winter oxygenated gasoline program, coordinated by the Office of Science and Technology Policy, Executive Office of the President (Zogorski *et al.*, 1997). Of 592 stormwater samples, MTBE was detected in 41 samples, or 6.9 percent of samples. MTBE was the seventh most frequently detected VOC, behind toluene (23.2%), total xylene (17.5%), chloroform (13.4%), total trimethylbenzene (12.4%), tetrachloroethene (8.0%), and naphthalene (7.4%). MTBE was detected in stormwater from eight of the sixteen cities: Atlanta, Baton Rouge, Birmingham, Colorado Springs, Denver, Dallas-Fort Worth, San Antonio, and Phoenix. Detected concentrations ranged from 0.2 µg/L to 8.7 µg/L, with a median detected concentration of 1.5 µg/L. Of the 41 detections, 34 (or 83%) were from samples taken during the winter months (from October to March) when MTBE would be used in greater quantities in some areas to meet federal air quality standards. In Phoenix, Colorado Springs, and Denver, three cities known to use oxygenated fuels, all detections were in the October-March season. The detection rate in these three cities was 40 percent (16 of 40 samples), with concentrations ranging from 1.0 to 4.2 µg/L and a median detected concentration of 1.5 µg/L. Detection in the other five cities (Atlanta, Baton Rouge, Birmingham, Dallas-Fort Worth, and San Antonio) is attributed to use of MTBE in gasoline as an octane enhancer. Note that the reporting limit for the analytical method used to monitor MTBE was reduced from 1.0 µg/L to 0.2 µg/L in 1994. It is likely that had the lower reporting limit been used throughout, the frequency of detection would be higher and the median detected concentration would be lower (Delzer *et al.*, 1996). The interagency report (Zogorski *et al.*, 1997) points out that the results of this monitoring might not be nationally representative, since none of the 16 monitored cities are located in California or the Northeast, where MTBE use is greatest.

American Water System Survey

The American Water System (AWS) of the American Water Works Company owns utilities in 23 States. In 1997 and 1998, AWS conducted its own survey of MTBE occurrence in source water. Two hundred surface water samples were taken from 92 sites in 12 States, and 1,349 ground water samples were taken from 342 wells in 17 States. Monitoring at all of the surface water sites and most of the ground water sites (from 270 out of 342) represented untreated source water. Where finished water was sampled, the treatment processes used (chlorination, pH adjustment, iron or manganese sequestration, fluoride addition) are not expected to affect MTBE concentrations. The method reporting limit was 0.5 µg/L (Gullick and LeChevallier, 2000).

In ground water, there were 136 MTBE detections (10 percent of samples) in 30 wells (8.8 percent of wells) in eight States. In surface water, there were 12 detections (6.0 percent of samples) at 8 sites (8.7 percent of sites) in three States. States with detections included Connecticut, Indiana, Massachusetts, Maryland, New Hampshire, New Jersey, New York, Pennsylvania, and West Virginia. In four States, detections exceeded 5 µg/L: One or two wells in Massachusetts had concentrations as high as 5.9 µg/L, three wells in New Jersey had concentrations between 1.5 µg/L and 11.6 µg/L, a stream and two wells near a leaking underground storage tank in Pennsylvania had samples with MTBE concentrations as high as 25.1 µg/L, 5.5 µg/L, and 14.2 µg/L, respectively, and one well in West Virginia had MTBE concentrations as high as 6.6 µg/L (Gullick and LeChevallier, 2000). '

14.3.3 Drinking Water Occurrence

Nationally representative data on MTBE occurrence in drinking water have been collected by large and small public water systems in accordance with EPA's first Unregulated Contaminant Monitoring Regulation (UCMR 1). For a complete description of the UCMR 1, see Chapter 2 and USEPA (2006c).

UCMR 1

UCMR 1 monitoring was conducted primarily between 2001 and 2003, though some results were not collected and reported until as late as 2005. As a List 1 contaminant, MTBE was scheduled to be monitored by all large CWSs and non-transient non-community water systems (NTNCWSs) and a statistically representative sample of small CWSs and NTNCWSs. The data presented in this report reflect UCMR 1 analytical samples submitted and quality-checked under the regulation as of July 2005. MTBE data were collected and submitted by 796 (99.5 percent) of the 800 small systems selected for the small system sample and 3,068 (99.0 percent) of the 3,100 large systems defined as eligible for the UCMR 1 large system census. MTBE data have been analyzed at the level of simple detections (i.e., ≥ minimum reporting level, ≥ MRL, or ≥ 5 µg/L). Currently, there is no health reference level (HRL) for MTBE.

Results of the analysis are presented in Exhibits 14-9 and 14-10. For small systems, MTBE detections were reported by 0.38% of public water systems (PWSs), representing 0.15% of the population served, equivalent to approximately 147,000 people nationally. Among large systems, 16 systems (0.52%) had detections, affecting approximately 750,000 people (0.34% of

the population served). MTBE was found much more frequently in ground water systems than in surface water systems.

A total of 26 MTBE detections were reported from 19 systems in 14 States. At the four surface water systems with detections, concentrations ranged from 8 µg/L to 33 µg/L, while at fifteen ground water systems with detections, concentrations ranged from 5 µg/L to 49 µg/L. Detections at concentrations higher than 20 µg/L were found at one small ground water system (49 µg/L), three large ground water systems (48 µg/L, 36 µg/L, and 33.2 µg/L), and one large surface water system (33 µg/L). California, Georgia, New Hampshire, New Jersey, and New York each had two systems with detections, while Connecticut, Illinois, Massachusetts, Missouri, New Mexico, Pennsylvania, South Dakota, Tennessee, and West Virginia each had one system with detections. When these results are combined with results from USGS's literature review (Delzer and Ivahnenko, 2003a), Random Survey (Grady, 2003), and Focused Survey (Delzer and Ivahnenko, 2003b), all but eight States (Alaska, Hawaii, Mississippi, Nebraska, Nevada, North Dakota, Utah, and Wyoming) have had at least one documented detection of MTBE in ambient or drinking water. Note that the MRL in these studies varied: in UCMR 1 the lowest reported detections were 5 µg/L, in the random and focused surveys they were 0.2 µg/L, and in the literature search they were 0.1 µg/L.

Exhibit 14-9: Summary UCMR 1 Occurrence Statistics for MTBE in Small Systems (Based on Statistically Representative National Sample of Small Systems)

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,268		--
Percent of Samples with Detections	0.09%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	N/A		--
Minimum Reporting Level (MRL)	5 µg/L		--
Maximum Concentration of Detections	49 µg/L		--
99th Percentile Concentration of Detections	49 µg/L		--
Median Concentration of Detections	12.7 µg/L		--
Total Number of PWSs	796		60,414
Number of GW PWSs	589		56,072
Number of SW PWSs	207		4,342
Total Population	2,758,082		45,414,590
Population of GW PWSs	1,937,327		36,224,336
Population of SW PWSs	820,755		9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs with Detections (≥ MRL)	3	0.38%	149
GW PWSs with Detections	3	0.51%	149
SW PWSs with Detections	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	4,150	0.15%	147,000
Pop. Served by GW PWSs with Detections	4,150	0.21%	147,000
Pop. Served by SW PWSs with Detections	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated separately for each population-served size stratum and then added to yield the national estimate of GW PWSs with detections (and population served) and SW PWSs with detections (and population served). For intermediate calculations at the level of individual strata, see EPA's UCMR 1 Occurrence Report, entitled "The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List."

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-Due to differences between the ratio of GW and SW systems with monitoring results and the national ratio, extrapolated GW and SW figures might not add up to extrapolated totals.

Exhibit 14-10: Summary UCMR 1 Occurrence Statistics for MTBE in Large Systems (Based on the Census of Large Systems)

Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	30,333	
Percent of Samples with Detections	0.08%	
99th Percentile Concentration (all samples)	< MRL	
Health Reference Level (HRL)	N/A	
Minimum Reporting Level (MRL)	5 µg/L	
Maximum Concentration of Detections	48 µg/L	
99th Percentile Concentration of Detections	48 µg/L	
Median Concentration of Detections	9 µg/L	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	3,068 1,376 1,692	
Total Population Population of GW PWSs Population of SW PWSs	223,101,388 53,170,587 169,930,801	
Occurrence by System	**Number**	**Percentage**
PWSs with Detections (≥ MRL) GW PWSs with Detections SW PWSs with Detections	16 12 4	0.52% 0.87% 0.24%
Occurrence by Population Served		
Population Served by PWSs with Detections Pop. Served by GW PWSs with Detections Pop. Served by SW PWSs with Detections	749,483 421,186 328,297	0.34% 0.79% 0.19%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½ HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs > ½ HRL, or by PWSs > HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½ HRL benchmark, or exceeding the HRL benchmark, respectively.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.

The following maps, based on UCMR 1 data, give an indication of the geographic distribution of MTBE occurrence in drinking water. Exhibit 14-11 shows the distribution of States with at least one detection. Exhibit 14-12 shows the relative frequency of detection in these States. Exhibit 14-13 shows the maximum concentration of MTBE at each system where it was detected. Measured concentrations were variable and the map does not distinguish regions that might have had consistently high contamination.

Exhibit 14-11: Geographic Distribution of MTBE in UCMR 1 Monitoring – States With At Least One Detection At or Above the MRL (≥ 5 µg/L)

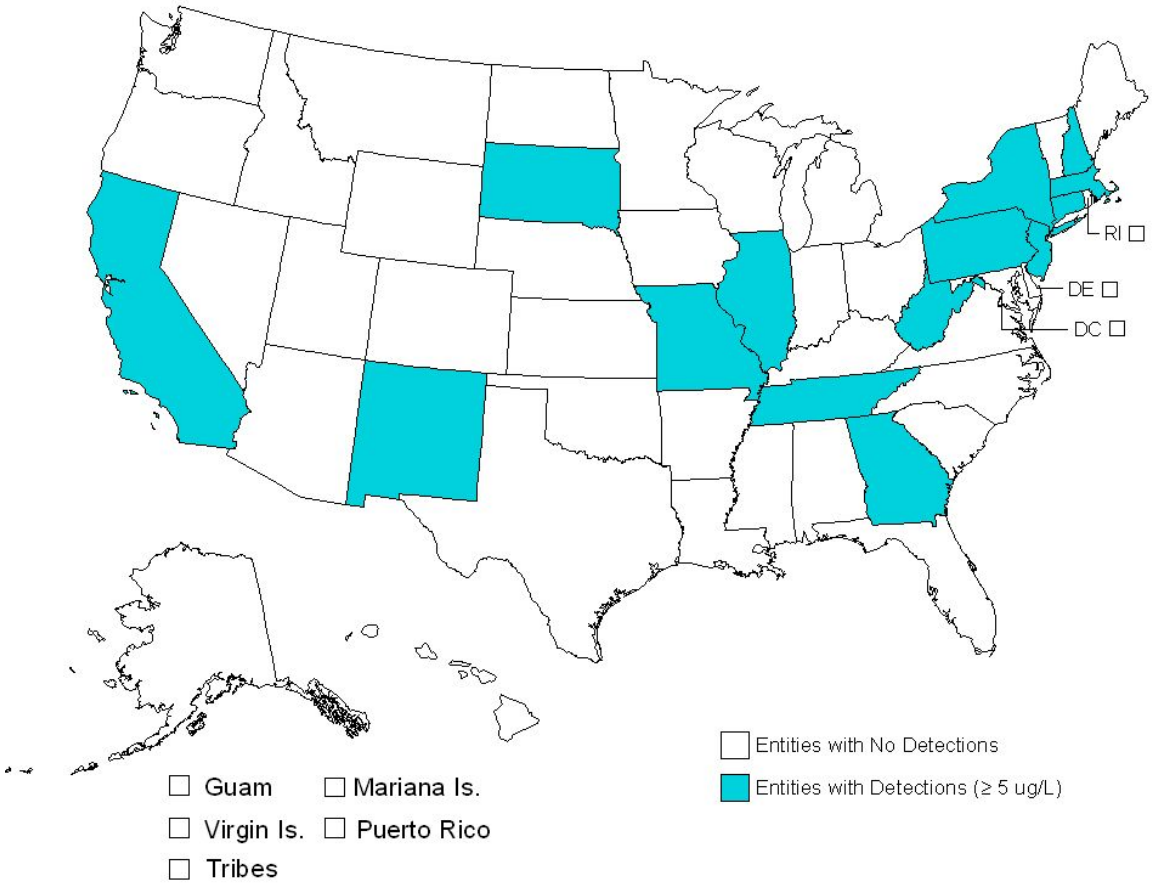
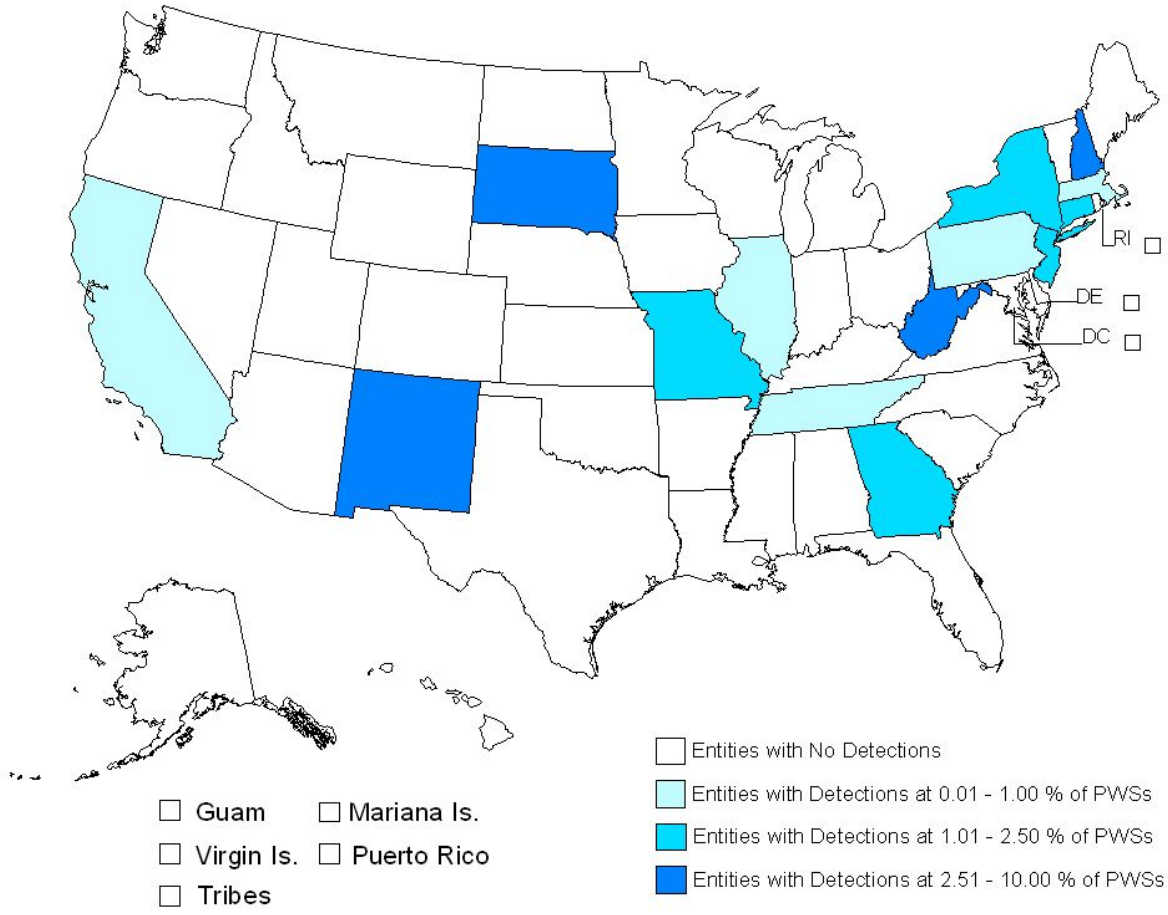


Exhibit 14-12: Geographic Distribution of MTBE in UCMR 1 Monitoring – Percentage of UCMR 1 PWSs With At Least One Detection At or Above the MRL (≥ 5 µg/L), By State



Note: This map depicts UCMR 1 results from both small systems and large systems. The statistical selection of UCMR 1 small systems was designed to be representative at the national level, but not at the state level. Therefore, this map should only be considered a rough approximation of state-level patterns of contaminant occurrence.

Exhibit 14-13: System-Level Geographic Distribution of MTBE in UCMR 1 Monitoring – Maximum Concentration at Each System with Detections

Compared with some national, regional, state, and local studies summarized in this report, the UCMR 1 occurrence data indicate low MTBE occurrence. When comparing UCMR 1 results to the results of other MTBE occurrence studies, several points should be considered. First, UCMR 1 is a survey of drinking water only. Many studies that report higher rates of MTBE occurrence (for example, USGS NAWQA studies and monitoring connected to UST investigations) are studies of ambient water.

Second, UCMR 1 was specifically designed to provide a statistically representative picture of national MTBE occurrence. In fact, UCMR 1 is the only study of MTBE in finished drinking water study that is both national in scope and statistically representative. (Among source water studies, the USGS Random Survey meets both criteria.) MTBE studies that target areas of known MTBE use or contamination, and/or have increased rates of sampling in such areas (e.g., the USGS Focused Survey and some state studies), are not representative, and likely overestimate MTBE occurrence.

Third, UCMR 1 sampling was undertaken in a specific timeframe: most samples were collected between 2001 and 2003 (with additional samples collected as late as 2005). Differences between UCMR 1 results and results of studies from the 1990s might be partially

attributable to actual changes in MTBE use and release over time. In addition, some state MTBE occurrence databases (e.g., California's) include historical results from contaminated wells that are no longer in use.

Finally, UCMR 1 used a minimum reporting level (MRL) of 5 µg/L. This is significantly higher than the reporting level used by most other studies, which was often the method detection limit. (All other things equal, the lower the MRL, the higher the rate of detections found and the higher the MRL, the lower the rate of detections.) For UCMR 1, EPA established MRLs for each volatile organic compound (VOC) by multiplying by 10 either the VOC's published detection limit, or 0.5 µg/L, whichever is greater (USEPA, 2000b; 64 FR 50556). EPA set MRLs approximately an order of magnitude higher than detection limits to ensure consistency, accuracy, and reproducibility of results in a study that involved analyses by many laboratories across the country, using multiple analytical methods. The threshold of 5 µg/L is also low enough to capture trends of interest. It is below EPA's advisory range for organoleptic (taste and odor) effects (20 µg/L to 40 µg/L), and below all primary drinking water standards for MTBE established to date by individual states (ranging from 10 µg/L to 240 µg/L – see Exhibit 14-22.)

When compared at a common threshold of 5 µg/L, occurrence is fairly consistent across the UCMR 1 and other studies. UCMR 1 found MTBE in 0.52% of large systems and 0.38% of small systems using the 5 µg/L MRL. At the same threshold, the USGS Survey of Drinking Water in the Northeast and Mid-Atlantic States (a statistically representative regional study focused on a region of high MTBE use, discussed below) found MTBE in 1.5% of samples from 2.0% of systems. The USGS Random Survey (a nationally representative survey of source waters for community water systems, discussed above) found that MTBE concentrations exceeded 5 µg/L in three of 934 tested source waters (0.3%). The American Water System's survey (a large but not necessarily representative sampling of source and finished waters, discussed above) found MTBE at concentrations above 5 µg/L in 1 out of 92 surface water sources (1.1%) and up to 8 of 342 wells (2.3%).

For further analysis of UCMR 1 results for MTBE, see the UCMR 1 occurrence analysis background report (USEPA, 2006c).

Summary Analysis of Combined Large and Small System UCMR Data

EPA found that 19 public water systems (0.49 percent of the 3,864 systems sampled) in 14 states (CA, CT, GA, IL, MA, MO, NH, NJ, NM, NY, PA, SD, TN, and WV) reported MTBE occurrence in drinking water. These 19 systems reported MTBE in 26 samples at or above the minimum reporting level of 5 µg/L, representing approximately 0.33 percent (or 754 thousand of 226 million) of the population served by the public water systems that sampled for MTBE. The average MTBE concentration among detections was 15.2 µg/L and the median concentration was 9.2 µg/L.

Community Water System Survey

The 2000 Community Water System Survey (CWSS) (USEPA, 2002a; 2002b) gathered data on the financial and operating characteristics of a random sample of community water systems nationwide. In addition, the Survey asked all "very large" community water systems, those that serve more than 500,000 people (a total of 83 systems), to provide monitoring results for five regulated compounds (arsenic, atrazine, 2,4-D, simazine, and glyphosate) and four unregulated compounds (radon, MTBE, metolachlor, and boron), including results from raw water at each intake and from finished water at treatment plants. EPA received responses from 58 systems. However, not all systems answered every question. Note that because reported results are incomplete, they are more illustrative than statistically representative.

Results of raw water monitoring are aggregated by type of intake. In raw ground water, 12 observations of MTBE occurrence were reported. In raw surface water, 27 observations of MTBE occurrence were reported (USEPA, 2002b).

Results of finished water monitoring are aggregated by system type. At systems primarily served by ground water, no MTBE observations were reported. At systems primarily served by surface water, 29 observations of MTBE occurrence were reported. At systems primarily served by purchased water, 3 observations of MTBE occurrence were reported (USEPA, 2002b).

The unpublished database from which the statistics above were drawn provides additional information. The 71 reported MTBE detections came from fifteen systems in eleven States (California, Georgia, Kentucky, New Jersey, New York, Ohio, Pennsylvania, Rhode Island, Texas, Virginia, and Wisconsin). All fifteen systems had raw water detections, four from ground water sources and 12 from surface water sources. (One Ohio system had detections in both surface and ground water sources.) Excluding two obvious outliers (most likely due to mislabeling of units), the highest reported concentration in raw water was 501 µg/L, from a ground water intake at a New York system that primarily uses surface water. Of 37 reported raw water detections (excluding outliers), 14 were of MTBE concentrations higher than 20 µg/L. Eleven of the fifteen systems reported finding MTBE contamination in finished water also. At one system the contaminated water came from a purchased source; at the other ten the contaminated water came from surface sources. Excluding one obvious outlier, the highest concentration in finished water was 50 µg/L, found twice at a California system served primarily by surface water. Of 31 reported MTBE detections in finished water (excluding the outlier), 4 were of concentrations higher than 20 µg/L. As noted above, because of incomplete responses and the low response rate, these survey results can not be considered statistically representative.

USGS Survey of Drinking Water in the Northeast and Mid-Atlantic States

USGS compiled and analyzed occurrence data for MTBE and other VOCs in finished drinking water in twelve Northeast and Mid-Atlantic States (Connecticut, Delaware, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont, and Virginia), a region of high MTBE use (Grady and Casey, 2001). State agencies supplied USGS with VOC data collected during 1993-1998 for 20 percent of the CWSs in the twelve-State area, which were chosen to be representative in terms of geography, source of

water, and system size. Delaware and Pennsylvania did not have any MTBE data, so the MTBE analysis was limited to 1,194 CWSs in ten States. After the trihalomethanes, MTBE was the most frequently detected VOC. Concentrations at levels ≥ 1 µg/L were found in 248 samples (4.8 %) from 84 CWSs (7.8%); concentrations at levels ≥ 5 µg/L were found in 82 samples (1.5%) from 23 CWSs (2.0%); and concentrations at levels ≥ 20 µg/L were found in 27 samples (0.5%) from 10 CWSs (0.8%). Extrapolating from these results, the study authors estimated that the number of CWSs in the 12-State area with MTBE concentrations of ≥ 1 µg/L, ≥ 5 µg/L, and ≥ 20 µg/L is approximately 820-890, 180, and 80, respectively.

At concentrations greater than 1 µg/L, MTBE was detected seven times more frequently in areas where MTBE is or has been added to gasoline as part of the oxygenated or reformulated fuel programs than in other areas, a difference that is statistically significant. Differences between MTBE concentrations inside and outside those areas, however, were not found to be statistically significant (Grady and Casey, 2001).

USGS Survey of Untreated Rural Self-Supplied Domestic Wells

As part of the NAWQA program, USGS studied the occurrence of MTBE and other VOCs in ground water from untreated rural self-supplied domestic wells between 1986 and 1999 (Moran *et al.*, 2002). These sources of drinking water are not subject to EPA drinking water regulations. At a reporting level of 0.2 µg/L, 30 out of 1,335 wells contained MTBE. Most of the contaminated wells were concentrated in the northeast (Pennsylvania, New Jersey, Connecticut and Massachusetts); the remainder were in Arkansas, Colorado, Georgia, and Illinois. The median detected MTBE concentration was 0.7 µg/L. The sample with the highest concentration, 30.2 µg/L, was the only sample that exceeded 20 µg/L.

Environmental Working Group (EWG) Report

In April 2005, the Environmental Working Group (EWG, 2005) released a report, *Like Oil and Water*, on their web page. In response to Freedom of Information Act requests, 29 State agencies submitted data to EWG. EPA informally evaluated the data posted by EWG to determine if this information might be useful in projecting state-wide occurrence. While EPA found the report interesting, the data as reported on the Web lacked some of the information needed to assess the representativeness and the quality of the data. For example, States submitted different time periods of monitoring data (e.g., Alaska submitted 7 months of data for 1 system during the 2000 timeframe and Illinois submitted data that spanned 1994 to 2003). States did not report monitoring results for every system. Also, the data do not indicate if the samples came from source water or finished water, or from ground water or surface water; also not reported are the analytical method used for analysis, the reporting level, the frequency of the sampling (e.g., annual, quarterly), the number of samples from each water system, and the number of non-detects.

14.3.4 Prominent Cases of MTBE Drinking Water Contamination

Several high-profile cases of MTBE contamination affecting drinking water are summarized here. These accounts are drawn from published studies and State agency reports

where available. In other instances, the information comes from news reports and other secondary sources.

South Lake Tahoe, California

South Tahoe Public Utility District (STPUD) was among the first public water suppliers in the State of California to become aware of an MTBE contamination problem (Bourelle, 1998a). According to Bourelle (1998a), STPUD first detected MTBE at the Tata Lane No. 4 well in February of 1997. Another source suggests that STPUD may have detected trace amounts of the chemical even earlier, as early as 1996 (Primedia, 2002). Bourelle (1998a) states that the first two wells were shut down in September 1997. The Tata Lane No. 4 well itself continued in operation with treatment by air stripping until concentrations rose and traces of MTBE were detected in finished water. The Tata Lane No. 4 well was finally shut down in July of 1998. In addition to these three contaminated wells, STPUD shut down seven uncontaminated wells by August of 1998 as a precautionary measure. At least one other well was operated at half-capacity to prevent pulling the contaminant plume closer (Bourelle, 1998a).

The California Department of Health Services (California DHS) maintains a listing of MTBE detections at California PWSs on the Internet (California DHS, 2005). According to California DHS, only active or inactive sources that have had at least two detections are listed. "Sources" include raw and treated well water and surface water, distribution system water, etc. As of May 2005, the database lists 143 detection records from 14 sources at South Lake Tahoe (or 13 wells, because raw and treated water from the "Gardner Mt. WTP" well are listed separately). Sample dates range from June 1996 to July 2005. The median concentration for all South Lake Tahoe records is 1.3 µg/L, with a maximum concentration of 68 µg/L detected at Tata Lane Well No. 4 in July of 1999. See Exhibit 14-14 for a summary of South Lake Tahoe results posted by California DHS (2005).

Exhibit 14-14: MTBE detections in wells of South Tahoe PUD

Source Name	Concentration (µg/L)		
	Min	Median	Max
Arrowhead Well 01 - Destroyed	2.5	2.9	3.7
Arrowhead Well 02 - Destroyed	1.2	1.4	1.8
Bakersfield Well – Raw	0.25	0.28	0.6
Blackrock Well 01 – Monitoring Well	1.8	1.8	1.8
Chris Ave Well	0.23	0.23	0.23
Clement Ave Well	0.5	0.5	0.5
Country Club Well (Angora 07)	0.5	0.65	1
Gardner Mt. Wtp - Raw	1.4	3.9	5
Gardner Mt. Wtp - Treated	0.5	1.2	2.3
Julie Well	0.22	1.3	2.2
South Y Center Well - Inactive	0.7	2.15	4.2
Tata Lane Well 02	0.5	0.6	1
Tata Lane Well 03	0.5	0.7	1.2
Tata Well 04	0.5	18	68

Source: California DHS, 2005.

Authorities identified six contaminant plumes originating from South Lake Tahoe gas stations as sources of MTBE contamination (Bourelle, 1998a). The Lahontan Regional Water Quality Control Board published periodic reports listing all known discharges of MTBE in the region, including those in South Lake Tahoe. The most recent report (Dodds, 2004), from April 2004, lists 22 USTs in South Lake Tahoe (27 in El Dorado county) with known MTBE discharges, plus an additional UST in South Lake Tahoe with a ground water MTBE detection but no confirmed discharge. Most USTs were located at gas stations. The report also lists 5 MTBE discharges in South Lake Tahoe labeled "Spills, Leaks, Investigations, and Cleanups," and one MTBE detection at a well. Most of the discharges and detections were first documented in 1998; the rest were first documented in 1999 (Dodds, 2004).

With 10 of 34 wells out of action and others operating at less than full capacity, water supply was 20 percent below normal in 1998. In the short term, STPUD responded to the shortage by implementing water usage restrictions, particularly during the summer (Bourelle, 1998a).

Soon after the wells were closed, STPUD filed suit against 31 companies, including large gasoline manufacturers and distributors and local gas stations (Bourelle, 1998b; Primedia, 2002). Between 1999 and 2002, the defendants agreed to settle for amounts totaling $69 million (Wood, 2002). STPUD netted $45 million from the settlements, $10 million of which had to be spent immediately to cope with the contamination. As of 2004, STPUD had installed an ozone treatment system and drilled several new, deeper wells. STPUD anticipated that water restrictions would remain in place indefinitely (Crofton, 2004).

Santa Monica, California

The City of Santa Monica has three well-fields. In August of 1995, the City discovered MTBE contamination in the Arcadia and Charnock wellfields. In 1996, the City shut down all production at the two contaminated well fields (LARWQCB and USEPA Region 9, 1998; 65 FR 16094). These seven wells (2 wells in Arcadia and 5 wells in Charnock) represented approximately 50 percent of the City of Santa Monica's drinking water supply (USEPA, 2005d). The California DHS database holds 39 records of MTBE ground water detections in the City of Santa Monica Water Division (California DHS, 2005). Basic statistics on concentrations detected at all Santa Monica wells listed by DHS are presented in Exhibit 14-15. The samples in the database date from 1995 and 1996.

Exhibit 14-15: MTBE Detections in Wells of the City of Santa Monica Water Division

Well Name	Concentration (µg/L)		
	Min	Median	Max
ARCADIA WELL 04	19.6	19.6	19.6
ARCADIA WELL 05	9.5	18.55	86.5
CHARNOCK WELL 13 - INACTIVE	44.4	133	490
CHARNOCK WELL 15 - INACTIVE	53.3	63.05	72.8
CHARNOCK WELL 16 - INACTIVE	3.1	3.1	3.1
CHARNOCK WELL 18 - INACTIVE	6.5	23.75	47.5
CHARNOCK WELL 19 - INACTIVE	8.2	300	610

Source: California DHS, 2005

The Los Angeles Regional Water Quality Control Board (LARWQCB) and EPA Region 9 identified a leaking UST system at one gas station as the source of MTBE at the Arcadia site (LARWQCB and USEPA Region 9, 1998). Investigation of USTs and gasoline pipelines near the Charnock wellfield revealed that 25 sites had released gasoline containing MTBE, and 12 of those releases had contaminated ground water (USEPA, 2005e).

The Southern California Water Company (SCWC), serving Culver City, also drew water from the Charnock Sub-basin. Though SCWC never detected MTBE in its two Charnock Sub-basin wells, it shut down both wells to prevent the migration of the contaminant plume (LARWQCB and USEPA Region 9, 1998).

Since 1996, the City of Santa Monica and the SCWC have replaced water from the contaminated wells by purchasing finished drinking water from the Metropolitan Water District of Southern California. The annual cost of this replacement water is approximately $3.2 million. Until 2000, the cost of replacing the water was paid by Shell, Chevron, and Exxon. From 2000 to 2005, under enforcement order by EPA and LARWQCB, 16 parties are responsible for funding the replacement water (USEPA, 2005d).

Glennville, California

In 1997, ground water in Glennville, California was contaminated with MTBE. The source was a leak from the town's only gas station (Weiser, 2004). Residential drinking wells had MTBE concentrations as high as 20,000 µg/L (65 FR 16094). Since 1997 the State has supplied water to Glennville (population c. 200) by truck. The town won a $500,000 settlement from oil companies toward the installation of a new community water system. State officials estimate that a new water system will cost $1.2 million (Weiser, 2004).

La Crosse, Kansas

La Crosse, Kansas, is served by several wells from a single aquifer. In May 1996, a resident complained of a strange odor in an irrigation well. Testing revealed that a nearby public well contained MTBE at a concentration of 200 µg/L (NFESC, 2000). In 1997, the Kansas Department of Health and Environment (DHE) found MTBE in excess of 1,000 µg/L at two

public water supply wells (Hatten, 2000). Authorities attributed the contamination to gasoline releases at three gas stations eight tenths of a mile away from the public wells (Hatten and Blackburn, 1999).

In September of 1997, a new treatment system was installed, consisting of two air strippers in series. Each tower removes 80 to 90 percent of MTBE. Together, they reduce concentrations MTBE from 200 - 600 µg/L in influent water to 24 µg/L or less in finished water (NFESC, 2000). This treatment system, and at least three others designed to remediate MTBE-contaminated water in other Kansas communities, were paid for out of the State's Underground Storage Tank Trust Fund (KDHE, 2000).

Whitefield, Standish, and North Windham, Maine

Not all cases of MTBE contamination leading to well closures are due to leaking underground storage tanks. Three high-profile cases of MTBE contamination caused by surface spills of reformulated gasoline occurred in Maine in 1998. These events contributed to Maine's withdrawal from the federal RFG program (described in Section 14.3.5, below).

In May 1998, MTBE contamination was discovered in a well serving an elementary school in the town of Whitefield. The MTBE concentration is variously reported as 500 µg/L, 800 µg/L, or 900 µg/L (Sullivan, 1998; Maine, 1998; NESCAUM, 1999a). State officials determined that the source of contamination was a spill of no more than 20 gallons in an area about 120 feet from the well where cars were parked on the grass (NESCAUM, 1999a). The school now filters the contaminated water for washing, and uses bottled water for drinking. At least one new well was drilled and installed (Sullivan, 1998).

Also in May 1998, MTBE was detected in private wells in Standish. Authorities attributed the contamination to a spill of 8 to 10 gallons of gasoline from a December 1997 car accident. The contamination affected a total of 24 private wells. Eleven wells were contaminated at levels above the State's 35 µg/L MTBE standard, requiring filtration; ten wells were contaminated at levels above 100 µg/L; two wells were contaminated at levels above 1,000 µg/L. The highest MTBE concentration, 6,500 µg/L, was recorded at the well nearest the accident site (Sullivan, 1998; Maine, 1998; NESCAUM, 1999a). The State responded to the contamination by removing 79 cubic yards of contaminated soil (NESCAUM, 1999a).

In the town of North Windham, MTBE was detected at two public water supply wells in concentrations ranging from 1 to 6 µg/L. Investigation revealed that the contamination originated from a new state-of-the-art gas station located 700 (or 900) and 1,100 feet, respectively, from the contaminated wells (Sullivan, 1998; NESCAUM, 1999a). A monitoring well near the gas station found MTBE at levels as high as 7140 µg/L (Sullivan, 1998). However, there was no leak in the double-walled UST and there was no evidence of vapor leaks. Investigators concluded that the source of the contamination must have been overfilling of the tank, and estimated that the amount of the spill was between 10 and 40 gallons (NESCAUM, 1999a). Extensive testing in North Windham following the initial detection found that 11 of 31 monitoring wells in the Windham aquifer had MTBE contamination above the detection limit (0.2 µg/L). North Windham now receives most of its water from Portland Water District (Nielson and Peckenham, 2000).

Merrimack River, Massachusetts

On the evening of January 28, 2000, a tanker truck rolled over in Lowell, Massachusetts, releasing a large amount of gasoline near the Merrimack River. According to a spokesman for the Lowell police, the amount spilled was "most of [the truck's] 11,900-gallon cargo" (Seymour, 2000). Responders attempted to contain that gasoline and vacuum it up. Warmer weather and the melting of river ice the next day allowed some gasoline into the river (Seymour, 2000).

Several downstream cities use the Merrimack River as a source of drinking water. Tewksbury and Lawrence temporarily closed their river intakes and drew water from reservoirs, but the treatment facility in Methuen remained open. The day after the spill, EPA and State officials found no MTBE in river water (Seymour, 2000). Later, MTBE was detected in the river water at elevated levels for a period of several days (65 FR 16094).

Pascoag, Rhode Island

In the summer of 2001, residents of Pascoag village (population 4000) in Burrillville, Rhode Island, complained of foul tastes and odors in their drinking water (Mello, 2001). On finding MTBE in the water, the Rhode Island Department of Health issued drinking and cooking restrictions in early September (RI Department of Health, 2002). The State provided a daily ration of ½ gallon of bottled water per person, installed an interim filtration system at the contaminated wells, and investigated possible alternative sources (Mello, 2001; USEPA, 2003b). In November, testing revealed that the new carbon filtration system successfully reduced MTBE levels from 1,100 µg/L in raw well water to non-detectable levels. Contamination in the distribution system fell to less than 10 µg/L (RI Department of Health, 2001). In January 2002, after the Pascoag system was linked to a new water supply in the neighboring village of Harrisville, the Department of Health lifted the drinking and cooking restrictions (RI Department of Health, 2002; USEPA, 2003b).

Investigation revealed that the source of the contamination was a leaking UST at a single gasoline station (USEPA, 2003b). By November 2001, the gas station had closed (Mello, 2001). In 2002, a $1 million grant from EPA's Leaking Underground Storage Tank Fund was used to remove the UST system and 2000 cubic yards of contaminated soil and to install a ground water remediation system. In June 2003, EPA announced that it would provide an additional $1 million. The funds would enable the Rhode Island Department of Environmental Management to install a second ground water treatment system that would include an experimental bio-reactor component, to be piloted for six months in collaboration with EPA's Office of Research and Development (USEPA, 2003b).

Dallas, Texas and Lake Tawakoni

On March 9, 2000, a ruptured 28-inch pipeline released approximately 600,000 gallons of reformulated gasoline in a pasture several miles northwest of Greenville, Texas. The spill drained into a creek approximately 28 miles upstream from Lake Tawakoni, a major reservoir used by the city of Dallas and other surrounding towns. On March 12, heavy rainfall washed the spill into the reservoir. Approximately 11,500 pounds of MTBE entered the reservoir by March

13. Monitoring at the time suggested that MTBE entered the reservoir as one "slug," rather than in a continuous stream (SRA, 2000).

Initial MTBE concentrations in the lake were as high as 6,000 µg/L. As the contamination spread through the lake, the highest concentrations were found to migrate along the banks, where the plume was not diluted by deep waters. At the first major intake, for the city of West Tawakoni, MTBE concentrations peaked at 740 µg/L on March 18. At the intake for the Cash Water Supply Corporation (Cash WSC), contamination peaked at 19 µg/L on March 20. On March 24, MTBE concentrations at the intakes for the cities of Commerce and Greenville peaked at 11 µg/L. By the time the plume reached the Dallas intake on March 25, detected concentrations did not exceed 1.3 µg/L. By April 19, detections throughout the reservoir were at or below 1.0 µg/L, and detections effectively ceased after May 30. Authorities attributed later sporadic detections of MTBE to the use of sport water craft on the lake (SRA, 2000).

The owner of the ruptured pipeline, Explorer Pipeline Company, responded to the spill under the supervision of EPA and State agencies. Explorer worked with the Sabine River Authority (SRA), which owns and operates the reservoir, to monitor the contamination in the lake and protect drinking water intakes. The responses of the various water customers on Lake Tawakoni varied, depending on their proximity to the point of contamination and the availability of alternative sources. The city of Dallas, which has rights to 80 percent of the water in the reservoir, shut off its intake from March 10 until August 16 and relied on alternative sources. According to SRA (2000), shutting down the large Dallas intake probably slowed the migration of the plume across the lake, giving others more time to respond. Explorer installed an aeration system on March 14 to try to volatilize the MTBE before the plume reached the first drinking water intake, but high MTBE concentrations forced the city of West Tawakoni to shut its intake on March 15. Explorer provided drinking water by truck to West Tawakoni from March 16 to March 24. Explorer also installed a carbon filtration system at the West Tawakoni treatment plant on March 22, which was found to successfully mitigate the MTBE contamination (45 µg/L at the time of installation). Cash WSC did not close its intake. The city of Commerce did not close the intake entirely, but restricted its use when the contamination was at its highest. Greenville shut down its intake on March 10 and relied instead on water from the city's private reservoir until April 25. Commerce and Greenville both installed aeration systems in late April as precautionary measures. Other water consumers on Lake Tawakoni, whose intakes were not contaminated, nevertheless were affected by the incident: they increased monitoring, considered contingency plans, and handled questions from the public (SRA, 2000).

14.3.5 The Experience of Representative States with MTBE

States vary widely both in the magnitude of the threat posed by MTBE to drinking water resources and the way they mobilize resources to handle the threat. Variation in MTBE occurrence is due in part to the different requirements on regions to meet standards of the Clean Air Act. Certain States are part of the Federal Reformulated Gasoline (RFG) or wintertime Oxyfuel programs, which require higher oxygenate levels in gasoline (roughly 11 to 15 percent MTBE by volume, when MTBE is used), while others just use MTBE as an octane-enhancer (with levels as high as 8 percent by volume). Still others have banned MTBE use entirely. Furthermore, even in States that have not banned MTBE, other oxygenates are sometimes preferred for meeting federal requirements. Notably, in the corn-producing Midwest, ethanol is

a particularly cost-efficient alternative. Other factors that influence nationwide heterogeneity in MTBE occurrence are the distribution of LUSTs, variations in the concentration of MTBE sold in gasoline in geographic regions, and the layout of the gasoline transportation network that allows contamination and mixing.

The following sections detail the experience of sixteen States with MTBE. The States chosen are those with "State Investigation Reports on MTBE" posted on EPA's Underground Storage Tank webpage (USEPA, 2004b). These States represent a variety of experiences with MTBE contamination. Not all States that would be expected to have the greatest impacts from MTBE (e.g., RFG States) are included. Information was gathered from available literature and web searches.

Arizona

Starting in 1989, Arizona required all gasoline sold in Maricopa County in wintertime to contain oxygenates (15 percent MTBE by volume, if MTBE was used). The requirement was extended to Pima County in 1990. Initially, MTBE was the most common oxygenate used to meet this requirement (80% MTBE v. 20% ethanol in Maricopa County). However, by 1993 ethanol was the preferred oxygenate for wintertime fuel (73% ethanol v. 27% MTBE in Maricopa County and 74% ethanol v. 26% MTBE in Pima County). By 1999, nearly 100% of wintertime gasoline in the two counties contained ethanol (ADEQ, 1999).

Phoenix and Maricopa County also participated in the federal RFG program until 1998, when they were permitted to opt out because the State had adopted more stringent standards of its own (USEPA, 2005b). Starting in 1997, Arizona required all gasoline sold in Maricopa County in the summer months to be Arizona Clean-Burning Gasoline (CBG) (requiring 11% MTBE by volume, if MTBE is used). Because of the relatively high vapor pressure of ethanol-containing gasoline, MTBE was consistently the preferred oxygenate used to meet summertime CBG requirements (ADEQ, 1999). However, due to ground water contamination concerns, in 2004 the governor of Arizona signed a bill banning MTBE in gasoline, effective January 1, 2005 (U.S. Water News Online, 2004; Arizona Revised Statutes 41-2122).

The Arizona Department of Health Services (ADHS) has established a health-based guidance level of 35 µg/L for MTBE in ground water. The level is not a regulatory or compliance standard and therefore is not enforceable (ADEQ, 1999).

According to information available to EPA, there are 8,119 active USTs in Arizona, and there have been 8,137 confirmed releases. Cleanups have been performed on 5,540 of those releases (68 percent) (USEPA, 2005f). According to Dahlen *et al.* (2003), the State is aware of over 9,000 USTs (presumably including inactive USTs), about 4,300 of which have attained LUST status. ADEQ (1999) does not track MTBE occurrences within its UST database, but it has begun monitoring MTBE at "corrective action sites." Approximately 65 percent of Arizona's population uses ground water as their principal drinking water source (ADEQ, 1999).

Dahlen *et al.* (2003) reviewed 417 Arizona LUST site files and conducted additional field research, including collecting more than 700 supplemental ground water samples. Sites with known MTBE contamination were preferred over others, and 97 percent of the leaks occurred

prior to the implementation of 1998 upgrade requirements. Dahlen *et al.* (2003) found that MTBE has been detected in Arizona ground water at levels above 20 µg/L in at least 54 cities, and that MTBE occurrence was relatively widespread in rural areas of the State as well. Among samples collected in source zones at gasoline-contaminated sites, MTBE was found in detectable concentrations (≥ 1 µg/L) in 71 percent of wells at 83 percent of sites. The median concentration was 330 µg/L, and the maximum concentration was 68,000 µg/L. Based on hydrological modeling and geographic information system (GIS) analysis, the study authors estimated that 6 percent of the approximately 10,000 municipal wells and 2.5 percent of the approximately 19,000 domestic wells in Arizona are in settings that make them vulnerable to LUST contamination.

In contrast, a joint monitoring effort by USGS and Arizona's State Water Quality Division in 1998 found no MTBE in 146 samples from the Upper Santa Cruz, Willcox, and Sacramento Valley Groundwater Basins (ADEQ, 1999). ADEQ reported that further ground water investigations were underway.

California

Gasoline in California has included MTBE in small quantities as an octane enhancer since the late 1970s. As in other States, MTBE use increased in 1992 when California entered into the wintertime Oxyfuel program. In 1994, the California Environmental Protection Agency (CAEPA, 1994) reported that Southern California was scheduled to join the federal RFG program in 1995, and that a Statewide program would require all California gasoline to meet stringent RFG standards by June 1, 1996.

In 1999 California established a secondary (nonregulatory) taste and odor threshold of 5 µg/L for MTBE in drinking water. Effective in 2000, California established a health-based primary maximum contaminant level (MCL) of 13 µg/L (California DHS, 2000). This value is also the public health goal (PHG) for the State. California PWSs are required to report detections of MTBE to the California DHS when concentrations are greater than 3 µg/L (California DHS, 2004a). According to California's annual compliance reports, there were no violations of the MCL in 2002 or 2003, and two violations (at two different systems) in 2004 (California DHS, 2002; 2003; 2004b).

MTBE was first detected in California drinking water in 1989 and 1990 around San Francisco. California DHS first required monitoring of MTBE in some PWSs as an unregulated contaminant starting in 1997. Since then, California DHS has kept a database of every reported source and sample tested for MTBE in the State. A number of papers have reviewed this data, including Deeb *et al.* (2003), Williams (2001), and Williams *et al.* (2004). California DHS provides public access to data on MTBE occurrence at sites with two or more detections (California DHS, 2005). However, the public database does not systematically differentiate surface water from ground water or source water from drinking water, and it does not describe the number of tests per site.

Exhibit 14-16 contains an analysis by Deeb *et al.* (2003) of California source water monitoring data obtained from California DHS, updated through January 1, 2002. These statistics suggest that surface water contamination is a greater problem than ground water

contamination, and that detections peaked in 1999. Deeb *et al.* (2003) suggest that the apparent decline in detections after 1999 could be due to watershed protection measures and other new regulations, or it might simply be an artifact of sampling patterns.

Exhibit 14-16: Detection of MTBE in California PWS Sources

	1995	*1996*	*1997*	*1998*	*1999*	*2000*	*2001*
Detections at Ground Water Sources							
3 < MTBE < 5 µg/L	1	0	1	4	8	5	7
5 < MTBE < 13 µg/L	0	0	0	3	4	5	5
MTBE > 13 µg/L	0	1	3	3	3	3	3
Total # detections	1	1	4	10	15	13	15
Total # detections > 5 µg/L	0	1	3	6	7	8	8
Total # sources sampled	89	1,666	2,289	3,151	3,208	2,868	5,248
% detection frequency	1.12%	0.06%	0.17%	0.32%	0.47%	0.45%	0.29%
Detections at Surface Water Sources							
3 < MTBE < 5 µg/L	0	1	1	3	6	5	2
5 < MTBE < 13 µg/L	0	2	5	3	5	3	4
MTBE > 13 µg/L	0	0	2	2	0	1	0
Total # detections	0	3	8	8	13	9	6
Total # detections > 5 µg/L	0	2	7	5	5	4	4
Total # sources sampled	4	96	176	197	228	251	300
% detection frequency	0.0%	3.1%	4.5%	4.1%	5.7%	3.4%	2.0%
Detections at Mixed/Unclassified PWS Sources							
Total # detections	0	0	0	0	0	0	0
Total sources sampled	15	121	162	221	262	248	389

Source: Deeb et al., 2003, using data from California DHS

Other analyses of California DHS data show similar patterns. Using the same data set but through the end of 2002, Williams *et al.* (2003) found that detection frequencies were significantly higher in surface water than in ground water. However, surface water detections tended to be at concentrations below State drinking water standards. Williams *et al.* (2003) presented evidence that despite increased monitoring between 1998 and 2002 in California, the rate of discovery of new instances of contamination leveled off or decreased. Also using California DHS data through 2002, Williams *et al.* (2004) reported that of 11,132 sites sampled for MTBE, 206 (or 1.9 percent) had at least one detection. The mean concentration of detections between 1998 and 2002 was 6.0 µg/L and the median was 3.0 µg/L. Of sites with detections, 9.2 percent of them had concentrations higher than 13 µg/L (the California MCL), and 6.3 percent had concentrations higher than 20 µg/L (the lower end of EPA's consumer advisory limit).

Of the 264 sources (i.e., wells) listed in the publicly available database (California DHS, 2005), 16% (41 sources) are currently designated as being "inactive," "abandoned," or "destroyed." These closed sources and their highest reported MTBE concentrations are listed in Exhibit 14-17. Note that occasionally sources are listed twice in the database, as when raw and finished water results are listed as separate entries.

Exhibit 14-17: Reported Closures of MTBE-Contaminated Water Sources in California (1989-2005)

County	System Name	Source Name	Maximum MTBE Conc. (µg/L)
BUTTE	Tank House	LPA REPORTED PRIMARY SOURCE - INACTIVE	4.5
	Cal-Water Service Co.-Chico	WELL 15-01 – INACTIVE	3.2
EL DORADO	South Tahoe PUD - Main	ARROWHEAD WELL 01 – DESTROYED	3.7
		ARROWHEAD WELL 02 – DESTROYED	1.8
		SOUTH Y CENTER WELL - INACTIVE	4.2
FRESNO	Musick Meadows #1	INACTIVE WELL	1.8
KERN	Caza Drilling California Inc	WELL 01 - INACTIVE	47
	CWS - Bakersfield	WELL 022-02 - RAW - INACTIVE	49.2
		WELL 064-01 - RAW - INACTIVE	12.3
		WELL 075-01 - RAW - INACTIVE	5.2
	Gaslite Mobile Home Park	WELL 01 - SYSTEM INACTIVATED	15.7
	Union Pacific Railroad Company	WELL 05 - INACTIVE	46
	Valley View Estates Mutual Water Co.	WELL 02 - INACTIVE	7.1
	Westside Industrial Center	WELL 01 - INACTIVE	0.74
LOS ANGELES	Calif State Polytechnical Univ - Pomona	WELL 01 - DESTROYED	2.8
	Los Angeles - City, Dept. of Water & Power	NORTH HOLLYWOOD WELL 17 - INACTIVE	3.5
		VERDUGO WELL 01 - INACTIVE	0.8
		VERDUGO WELL 02 - INACTIVE	13
	Santa Monica-City, Water Division	CHARNOCK WELL 13 - INACTIVE	490
		CHARNOCK WELL 15 - INACTIVE	72.8
		CHARNOCK WELL 16 - INACTIVE	3.1
		CHARNOCK WELL 18 - INACTIVE	47.5
		CHARNOCK WELL 19 - INACTIVE	610
	SCWC - Culver City	SENTNEY WELL 13 - ABANDONED	3.4
MERCED	Foster Farms Chicken Livehaul	WELL #2 - DESTROYED	2.71
MONTEREY	CWSC Salinas	13-02 GAC VESSEL -D- 25% - DESTROYED	4.81
		WELL 001-04 - DESTROYED	120
		WELL 013-02 - INACTIVE	400
NEVADA	Truckee-Donner PUD, Main	NEW DONNER CREEK WELL - INACTIVE	4.9
ORANGE	Southern Calif WC - Yorba	CONCERTO 01 - INACTIVE	40.9

County	System Name	Source Name	Maximum MTBE Conc. (µg/L)
	Linda		
PLUMAS	Quincy Community S.D.	WELL 01 - NORTON - DESTROYED	3.1
SACRAMENTO	Fruitridge Vista Water Company	WELL 11 - INACTIVE	26
SAN BENITO	Earthbound Farms	WELL 02 - MTBE & CL2 TREATMENT-INACTIVE	36
		WELL 02 - RAW - INACTIVE	25
SAN BERNARDINO	Sheep Creek Water Company	WELL 01 - DESTROYED	5.5
SAN DIEGO	Crystal Clear Water Company	WELL 01 - INACTIVE	10
SAN FRANCISCO	Presidio of San Francisco	WELL 06 - ABANDONED	23
		WELL 13 - ABANDONED	500
SANTA CLARA	Loma Prieta JUSD-Loma Prieta School	WELL 01 - INACTIVE	6.6
VENTURA	Calleguas Municipal Water District	FAIRVIEW ASR WELL - INACTIVE	0.59
YUBA	Cal-Water Service Co.-Marysville	WELL 03-01 - INACTIVE	234.1

Source: California DHS (2005)

A study at the Lawrence Livermore National Laboratory (Happel *et al.*, 1998) reported that 78 percent of 236 monitored LUST sites in California had detectable levels of MTBE, 74 percent had concentrations exceeding 5 µg/L, and 70 percent had concentrations greater than 20 µg/L. The study authors estimated that the number of MTBE-contaminated sites in the State is upwards of 10,000.

Cook *et al.* (2002) summarize data from multiple studies of MTBE occurrence in Santa Clara County ground water. One study analyzed drinking water from 51 domestic wells within a half-mile of LUST sites and found 4 detects, all at concentrations less than 10 µg/L. Another study sampled 104 drinking water wells every six months starting in 1999, and by the end of 2001, had not detected MTBE above the reporting limit of 3 µg/L. Although drinking water sources in Santa Clara County have not reported high levels of MTBE, ground water tests at LUST sites in the county have. Of 432 active LUST sites that tested for MTBE in 2001, 87 percent detected concentrations above 3 µg/L. Of those, 51 had concentrations exceeding 10,000 µg/L. A study by Tulloch (2000) found that of 16 locations in Santa Clara County that reported increasing trends in MTBE occurrence at significantly high levels, 13 had unreported leaks of gasoline that occurred after upgrading the storage tank. The report concluded that even after repairing and upgrading tanks, there can still be significant releases of fuel to soil and ground water.

In March 1999, Governor Gray Davis issued an executive order for the three-year phase-out of MTBE, making California the first State to officially ban the chemical. The order was codified by California SB 989, which also required refiners to submit quarterly reports on MTBE use and supply (NCSL, 2000). Because of complications, the ban was delayed beyond the

original December 31, 2002 deadline. California's phaseout was complete on December 31, 2003 (USEPA, 2004c).

Connecticut

Widespread use of MTBE as an octane booster in Connecticut started in the mid-1980s. Two parts of the State used MTBE in elevated concentrations when they were required to participate in the wintertime Oxyfuel program from 1992 to 1999. The entire State began participating in the Federal RFG program in January 1995. In 2000, approximately 95 percent of RFG gasoline sold in Connecticut used MTBE as the oxygenate (Connecticut DEP, 2000).

MTBE was first discovered in Connecticut's drinking water wells in 1987. In 1987, the Connecticut Department of Public Health (Connecticut DPH) established an action level of 100 µg/L for MTBE in drinking water. In March of 1999, Connecticut DPH lowered the action level to 70 µg/L. As of 2000, the Connecticut Department of Environmental Protection (Connecticut DEP) had found MTBE at concentrations greater than the DPH action level in 236 water supply wells, of which only 4 were public. Fifty-one PWSs and many more private wells had detected MTBE at concentrations less than the action level (Connecticut DEP, 2000).

After 1995, when the percentage of MTBE used in gasoline throughout the State increased to conform to federal RFG standards (typically from 3 to 11 percent by volume), the fraction of drinking water wells in the State that detected trace levels of MTBE increased significantly. Between 15 and 30 percent of all drinking water wells tested by Connecticut DEP from 1995 to 2000 contained concentrations of MTBE between 0.5 and 10 µg/L. Connecticut DEP considers contaminated rain or runoff as likely sources of low-level contamination, while releases from underground storage tanks account for more than 90 percent of wells exceeding the action level (Connecticut DEP, 2000).

A report by the Northeast States for Coordinated Air Use Management (NESCAUM, 1999a) provides more details on MTBE detections in Connecticut PWSs between 1997 and 1998. NESCAUM reports that 80 percent of the 607 CWS and 33 percent of the 647 NTNCWS in the State tested for organic contaminants during that time period. Of those tested in 1997, 30 PWSs detected MTBE at some level, and four detected concentrations above 10 µg/L. The maximum concentration was 210 µg/L. Of PWSs tested in 1998, 45 detected MTBE at any level, and 13 detected concentrations above 10 µg/L. Four systems had concentrations greater than 100 µg/L: 17,000 µg/L in Brookfield, 3,982 µg/L in Wilton, 400 µg/L in Durham, and 240 µg/L in Salem (NESCAUM, 1999a).

Connecticut Public Act 00-175, enacted on July 1, 2000, called for a ban on the sale and use of MTBE in the State and required Connecticut DEP to develop a plan to implement the ban (Connecticut DEP, 2004). The ban of MTBE in Connecticut began on January 1, 2004, the same date that New York's ban took effect (USEPA, 2004c).

Florida

Florida is not subject to federal RFG or Oxyfuel requirements. According to the Florida Department of Environmental Protection (Florida DEP), most gasoline sold in the State contains

3 to 8 percent MTBE (Florida DEP, 2004a). Florida started monitoring for MTBE as an unregulated contaminant monitoring in PWSs in the early 1990s (Florida DEP, 2004b) and started monitoring for MTBE in ground water at petroleum-contaminated sites in 1990 (Florida DEP, 2004a).

The Florida DEP data base (Florida DEP, 2004c) presents MTBE samples taken by the Florida Drinking Water Program between January 1993 and March 2000. The data set includes 8,439 samples from 1,692 PWSs. The data record 428 detections of MTBE at 20 PWSs (19 community water systems and 1 NTNCWS). The rate of detection is approximately 5.1 percent for samples, and 1.2 percent for systems. Of the detections, 101 (from 5 PWSs) ranged between 5 and 20 µg/L, and 3 (from 3 PWSs) exceeded 20 µg/L. The rest (324) were below 5 µg/L. On its webpage, Florida DEP (2000b) states that only two PWSs exceeded 20 µg/L (166 µg/L and 104 µg/L, both entry-point samples), but the database identifies a third PWS with a 41 µg/L plant sample. All of the detections were in ground water. Exhibit 14-18 presents the Florida MTBE detection data broken down by sample type. The detection rate of MTBE in treated drinking water (e.g., at the entry point or in the distribution system) was very low (< 0.5 percent).

Exhibit 14-18: MTBE Monitoring Results at Florida PWSs, Organized by Sample Type

Sample Type	Total Samples	Sample Detects	Percent Detects	Statistics for Recorded Detects		
				Minimum	Median	Maximum
Check	73	0	0.0%	-	-	-
Composite	373	1	0.3%	0.28	0.28	0.28
Distribution	1506	0	0.0%	-	-	-
Entry Point	3566	10	0.3%	0.82	2.80	166.00
Plant	43	3	7.0%	1.30	2.90	41.00
Quarterly	1814	258	14.2%	0.10	1.66	19.30
Raw	1317	154	11.7%	0.21	0.70	13.00
Special	47	2	4.3%	0.70	0.71	0.72
Total	8739	428	4.9%	0.10	1.39	166.00

Source: Florida DEP, 2004c

Hawaii

Hawaii is not required to sell or distribute either RFG or Oxyfuel, and consequently, has low levels of MTBE in its gasoline supplies. The Hawaii Department of Health conducted an inquiry in 1997 to determine the extent to which MTBE was used in the State's gasoline formulation. The inquiry revealed that a major refinery on Oahu had used MTBE in the past, and that this gasoline had likely been distributed to all major gasoline retailers and defense facilities in the State. There were also cases where gasoline containing MTBE had been

imported into the State. Subsequent testing revealed a number of LUST sites with MTBE-contaminated soil and ground water (Hawaii DOH, 1998).

Based on these findings, Hawaii DOH amended its UST policy to require monitoring for MTBE at petroleum release sites. Hawaii DOH established an action level of 20 µg/L for MTBE in aquifers used for or potentially used for drinking water, and 202 mg/L (202,000 µg/L) in non-drinking water aquifers to protect aquatic life (Hawaii DOH, 1998).

Hawaii DOH (1998) did not provide specific information on MTBE detections, or on the impact of MTBE on Hawaii's drinking water resources. A single Hawaii DOH press release, dated November 23, 2004, documents the detection of MTBE in a drinking water well at a concentration of 1.2 µg/L (Hawaii DOH, 2004). This concentration is below that typically reported by State health departments.

Idaho

Idaho has never participated in federal RFG and Oxyfuel programs. Like other States, it has had low levels of MTBE in gasoline since 1979 as a lead replacement.

Wicherski (1999) studied the occurrence of MTBE in Idaho ground water at 100 LUST sites with known petroleum contamination. MTBE was found at 50 percent of sites with recent gasoline releases (less than 5 years old), but only at 30 percent of sites with older gasoline releases. MTBE was found at no diesel release sites. The mean concentration of recent gasoline releases was 2,271 µg/L, and 312 µg/L for older releases. The maximum concentration, 15,900 µg/L, was found at a recent release site. MTBE was found at concentrations between 5 and 20 µg/L at seven sites, and at concentrations greater than 20 µg/L at 25 sites. Thus most contamination of ground water was either negligible (< 5 µg/L) or significant (> 20 µg/L), with few concentrations in between. No information was provided on the impact of drinking water wells by pollution of ground water at these sites (Wicherski, 1999).

Iowa

As of 2000, Iowa was not required to participate in federal RFG program. According the Iowa Department of Agriculture and Land Stewardship and the Petroleum Marketers of Iowa, MTBE was neither used nor sold in Iowa at that time. However, MTBE was commonly used in the late 1970s and 1980s as an octane enhancer. In response to concern that MTBE could be harming ground water, in 1999 the Iowa legislature required that all soil and ground water samples collected from LUST sites after July 1, 1999 be analyzed for MTBE (Iowa DNR, 2000).

A report by the Iowa Department of Natural Resources (IDNR, 2000) describes the results of MTBE sampling at LUST sites between July 1, 1999 and December 17, 1999. Of 2,569 ground water samples collected during this period, about 32 percent of them reported MTBE above the quantitation limit of 15 µg/L. Almost 29 percent of samples had MTBE levels above 20 µg/L. Approximately 55 percent of sites had at least one ground water sample with an MTBE concentration greater than 20 µg/L. The mean concentration of MTBE in ground water samples with detections above the quantitation limit was 613 µg/L and the median was 81 µg/L. The mean concentration of MTBE in all ground water samples was 200 µg/L and the median

was 5 µg/L. In soil, MTBE was detected in 60 percent of samples at 62 percent of sites. In addition, 53 water samples were collected from various "receptors" (e.g., drinking water wells, plastic water lines), of which only five samples had concentrations greater than 15 µg/L. One sample from a private drinking water well had an MTBE concentration higher than 20 µg/L (23.7 µg/L) (IDNR, 2000).

The same report also presented the results of a 1999 Iowa DNR study of 235 PWSs with wells in vulnerable bedrock (e.g., fractured systems). Only eight out of the 1048 samples (0.76 percent) contained MTBE, all of them at levels below the quantitation limit of 15 µg/L. In addition, the State is aware of MTBE detections at three other municipal water systems, all in northwest Iowa. Ida Grove has had low-level detections since 1997, reaching a maximum concentration of 12.0 µg/L in September 1998 (post-treatment, pre-blending). Galva detected MTBE at a concentration of 18 µg/L in 1996. Alford detected a maximum of 63 µg/L in 1994. In part because of MTBE contamination, the latter two cities have abandoned their wells and switched to other regional water sources (Iowa DNR, 2000).

The same bill that required MTBE monitoring at LUST sites also established a limit of 2 percent MTBE by volume in gasoline sold in the State, starting February 1, 2000 (Iowa DNR, 2000). Effective on July 1 of 2000, Iowa lowered the cap on MTBE ban in gasoline to 0.5 percent by volume (USEPA, 2004c).

Maine

Maine voluntarily chose to participate in the Federal RFG gasoline program. Seven Maine counties participated from 1995 until the State was officially allowed to "opt out" in 1999 (Maine DEP, 2005). Around 1997 the Maine legislature adopted an enforceable drinking water standard for MTBE of 35 µg/L–at the time the most stringent standard in any State–and the Maine Bureau of Health has monitored for MTBE in PWSs (Maine, 1998; Sullivan, 1998). At the same time, the Maine Department of Environmental Protection (Maine DEP) adopted an even more conservative action level of 25 µg/L for MTBE remediation (Maine, 1998; Sullivan, 1998).

The first known case of MTBE contamination in Maine occurred in 1984. A farmer's storage tank leaked gasoline containing 3 percent MTBE (by volume) and contaminated several local wells. Two years after the leak was discovered, levels of MTBE were still detected in local ground water at concentrations greater than 10,000 µg/L (Garrett *et al.*, 1986 as cited in NESCAUM, 1999a). In 1998, three incidents involving spills or tank overfills caused contamination of public and private wells with MTBE (described in Section 14.3.4, above). These incidents were highly publicized and created a sense of urgency about non-LUST sources of contamination.

In response to rising concern, Maine's governor directed State agencies to survey of MTBE occurrence in the State's drinking water resources. All of the State's public water systems and 1000 randomly selected private water supplies were to be sampled. The final study, published in 1998, used samples from 793 of the State's 830 nontransient public water supplies and 951 residential water supplies. Of the public water supplies, 125 sites (16 percent) had MTBE detections. Of these sites, 6.1 percent had MTBE concentrations of 1.0 µg/L or higher;

no detections exceeded 35 µg/L. Of the household water supplies, 150 (15.8 percent) had MTBE detections. Of these sites, 7.7 percent had concentrations of 1.0 µg/L or higher, and 1.1 percent had concentrations greater than 35 µg/L. Extrapolated Statewide, these findings suggest that approximately 1,400 - 5,200 private wells have MTBE contamination in excess of 35 µg/L Statewide. A comparison of results in different parts of the State showed that community water systems were 1.7 to 4.1 times more likely to be contaminated in areas of RFG use, depending on population density, and that private water supplies in such areas were 1.3 to 2.0 times more likely to be contaminated (Maine, 1998). It was on the basis of this study that Maine successfully petitioned to "opt out" of the federal RFG program in 1999 (Maine DEP, 2005).

In 2000, the Maine legislature adopted a goal of eliminating MTBE in gasoline by January 1, 2003 (Maine DEP, 2005). In April of 2004, a bill was adopted that will phase-out MTBE use by January 1, 2007, and cap concentrations in gasoline at 0.5 percent by volume (USEPA, 2004c). Ironically, in 2004 MTBE levels in Maine gasoline rose for the first time since 2000. State officials were unsure of the cause, but guessed that it might be due to a shift of regional refiners' low-MTBE gasoline stocks to Connecticut and New York, where bans were already in effect (Maine DEP, 2005).

Maryland

Use of MTBE in significant quantities in Maryland began in 1995, when major metropolitan parts of the State were required to participate in the RFG program (MDE, 2002). In 2001, a task force estimated that approximately 220 million gallons of MTBE are consumed each year in Maryland (Maryland Task Force, 2001).

While Maryland has no health-based limit for MTBE exposure, the State considers concentrations of 20 µg/L or more in water a trigger for treatment or replacement. In addition, MTBE concentrations at or above the action level of 10 µg/L trigger an investigation to find the source of the contamination (Maryland Task Force, 2001).

The Maryland Department of the Environment (MDE) started sampling for MTBE in non-transient PWSs in 1995. By 2002, MDE had tested 1,203 PWSs, of which MTBE was detected in 116 (9.6 percent). Thirteen systems (1.1 percent) had detections over 20 µg/L (MDE, 2002). Surface water systems, which serve 68 percent of Maryland residents, were hardly affected at all: Only 2 of the 99 PWSs with detections by 2001 were surface water systems, and neither had MTBE concentrations above 2 µg/L (Maryland Task Force, 2001). In 1999, MDE started sampling for MTBE in private wells (Maryland Task Force, 2001). By 2002, LUST site investigations had identified 338 domestic wells contaminated with MTBE (MDE, 2002). Deeb *et al.* (2003) ran a query of the Maryland Water Supply Database and gathered several statistics of occurrence. The results of this query are presented in Exhibit 14-19.

Exhibit 14-19: Detection of MTBE in public water supply systems in Maryland

Occurrence Characteristics	*1995*	*1996*	*1997*	*1998*	*1999*	*2000*	*2001*
3 < MTBE < 5 µg/L	0	2	17	19	15	27	32
5 < MTBE < 20 µg/L	2	7	10	8	5	5	8
MTBE > 20 µg/L	2	3	3	3	4	5	3
Total # detections > 3 µg/L	4	12	30	30	24	37	43
Total # detections > 5 µg/L	4	10	13	11	9	10	11
Total # systems sampled	940	596	555	659	388	384	323
% Detection Frequency (> 3 µg/L)	0.4%	2.0%	5.4%	4.6%	6.2%	9.6%	13.3%
% Detection Frequency (> 5 µg/L)	0.4%	1.7%	2.3%	1.7%	2.3%	2.6%	3.4%

Source: Deeb et al., 2003

As of 2001, Maryland had taken no significant action to ban or phase out the use of MTBE in gasoline (Maryland Task Force, 2001).

Michigan

As of 2002, Michigan did not participate in either federal fuels program. The presence of MTBE in conventional gasoline was monitored by the State's motor fuel quality program. Approximately 1800 samples were tested each year, though not in a statistical framework. Between 1994 and 1998, the percentage of gasoline samples containing MTBE in concentrations greater than 1 percent by volume declined from 40 percent to 13 percent. In 2000, 13 percent of samples contained MTBE in concentrations greater than 1 percent by volume, with an average concentration of 3 percent by volume, which suggests an estimated annual consumption of over four million gallons of MTBE in Michigan. In 2001, 17 percent of samples contained MTBE in concentrations greater than 1 percent, with an average concentration of 4.75 percent, suggesting estimated Statewide use of over 38 million gallons of MTBE (MDA, 2002).

Residual MTBE from pipelines was an additional source of MTBE in Michigan gasolines, in volumes as high as 2 percent. In 2000 and 2001, the State's motor fuel quality program found MTBE in 45 percent and 34 percent, respectively, of nominally non-blended gasoline samples (MDA, 2002).

According to the Michigan Department of Agriculture (MDA, 2002), the State's Department of Environmental Quality (MDEQ) has established a health-based MTBE limit of 240 µg/L in drinking water, and an aesthetic threshold of 40 µg/L. MDEQ's Surface Water Quality Division found no MTBE in a preliminary investigation of surface waters. MDEQ's Drinking Water and Radiological Protection Division found no MTBE detections in any major water supply, but did report isolated cases of MTBE in private wells near to LUST sites (MDA, 2002).

In 2000, Public Act 206 declared a ban on MTBE in Michigan, effective June 1, 2003. Michigan's single internal source of MTBE, a Detroit-area refinery that supplies approximately one quarter of the State's gasoline, planned to shut down its MTBE processor on October 1, 2002, and estimated that its MTBE stocks would be depleted by November 1, 2002 (MDA, 2002).

Missouri

Four Missouri counties and the city of St. Louis began participating in the Federal RFG program in 1999. Ethanol is the favored oxygenate in Missouri; MTBE is only used in approximately three percent of the reformulated gasoline sold in the St. Louis area (MDNR, 2004a).

Missouri has established three action levels for MTBE in drinking water. The limit for long-term exposure (equivalent to an MCL) is 20 µg/L. The limit for temporary exposure, such as while a PWS is searching for an alternate supply, is 400 µg/L. For acute exposures, 1000 µg/L is considered unsafe to drink for any length of time (MDNR, 2004a).

The State of Missouri began testing for MTBE at LUST sites in 1992 and in public water supplies in 1995 (MDNR, 2004a). Between 1992 and April of 2004, the State of Missouri found MTBE in 46 public and private drinking water wells at 30 currently active sites. These include 15 wells at 13 public supplies, with MTBE concentrations ranging from 1.48 µg/L to a high of 604 µg/L. MTBE concentrations at twelve of the wells peaked at less than 15 µg/L. Of 31 private wells, 25 had MTBE concentrations higher than 20 µg/L. The highest concentration in a private well was 17,000 µg/L (MDNR, 2004b).

As of 2000, the Missouri Public Drinking Water Program counted 1,444 CWSs and 241 NTNCWSs in the State (PSTIF, 2000). Based on those numbers, the 13 MTBE-contaminated PWSs represent less than 1 percent of Missouri public water systems.

In July of 2002, Missouri adopted a partial ban of MTBE, capping concentrations of gasoline sold or stored in the State at 0.5 percent by volume. The cap goes into full effect on July 31, 2005 (USEPA, 2004c).

Nevada

MTBE has been used in some premium fuels in Nevada since 1979. Both Las Vegas and Reno began using oxygenated fuels in 1989 to reduce wintertime air pollution, even before the federal program began in 1992 (NDEP, 1998). In early years, a combination of MTBE and ethanol was used to meet the oxygenation goals. However, by 2001, both metropolitan areas were using only ethanol to fulfill their oxygenate requirement (USEPA, 2001b).

In 1998, the Nevada Department of Environmental Protection (NDEP) set an interim action limit of 20 µg/L for MTBE in ground water at sites "in close proximity to receptors [e.g., people or fauna] and/or sensitive environments." At sites with "incomplete exposure pathways," the limit is 200 µg/L (NDEP, 1998).

New Hampshire

Four counties in southeast New Hampshire voluntarily opted into the Federal RFG program. Their participation began in 1995 (NHDES, 2002). In 2000, the NHDES compared the composition of gasoline in the six counties not required to have RFG to that in the four counties selling RFG. In the four-county RFG program area, all 40 samples met the RFG oxygen content requirement (2 percent). The MTBE content in these 40 samples ranged from 3.9 percent to 14 percent by volume. In the six-county area, they found that 5 percent of gasoline samples (7 of 140) contained enough MTBE and/or other oxygenates to qualify as RFG on the basis of oxygen content. An additional 18 percent (25 samples) had higher than expected levels of MTBE (4% to 10% by volume). In general, samples with elevated oxygen content were samples of mid- and higher grade gasoline. Other commonly detected oxygenates included tertiary-amyl methyl ether (TAME; 126 of 140 samples) and ethyl tertiary-butyl ether (ETBE; over 70 samples). A few samples also contained di-isopropyl ether (DIPE) and tertiary butyl alcohol (TBA), but none contained ethanol or methanol (NHDES, 2000a).

In 1993, New Hampshire began requiring all 1125 community and non-transient PWSs to monitor for VOCs. However, private State-certified labs were not required to test for MTBE until 1998. By March 1999, the State was aware of MTBE detections at 195 systems, including 171 active systems. Thirty systems (24 active) had detections greater than 5 µg/L, and 9 systems (4 active) had detections greater than 20 µg/L (NESCAUM, 1999a). In May of 2000, the State of New Hampshire lowered its drinking water standard for MTBE from 70 µg/L to 13 µg/L (NHDES, 2000b).

In 2000, 27 percent of private well samples analyzed in New Hampshire contained MTBE, and 4 percent exceeded the State standard of 13 µg/L. First-time detections of MTBE in public water supplies in 2000 were three to four times higher than pre-1995 levels. Also in 2000, NHDES found that 16.8 to 23.2 percent of the public water supplies in the four-county RFG area tested positive for MTBE, compared to 1.8 to 8.8 percent of public drinking water supplies in other areas of the State. Based on findings like these, New Hampshire petitioned EPA in 2001 for permission to opt out of the RFG program (NHDES, 2002).

In May of 2004, New Hampshire enacted a partial ban that caps MTBE concentrations in gasoline at 0.5 percent by volume. The ban will take place on either January 1, 2007, or 6 months after the State gains Federal approval to opt out of the RFG program, whichever date is later. New Hampshire's ban also applies to other gasoline ethers and TBA (USEPA, 2004c). In 2005, New Hampshire confirmed January 1, 2007 as the effective date of the ban (New Hampshire, 2005).

New Jersey

Twenty-one counties in New Jersey began participating in the wintertime Oxyfuel program in late 1992, and the entire State joined the Federal RFG program in 1995. The oxyfuel program ended in 1995 for the southern counties (the Philadelphia metropolitan area) and in 1999 for the northern counties (the New York/New Jersey metro area). In New Jersey, as in most other northeastern States, MTBE was and is the oxygenate most frequently used to meet oxyfuel and RFG requirement. For at least 20 years, MTBE has also been used to boost octane

in conventional gasoline, in concentrations of 2 to 8 percent in premium grades, and somewhat less in regular grades (New Jersey DEP, 2003).

Development of a health-based standard for MTBE in drinking water in New Jersey began in the mid-1980s, when concentrations as high as 81 µg/L were discovered in public water supplies. The process culminated with the establishment of a Maximum Contaminant Level (MCL) of 70 µg/L in 1996. The MCL was based on the finding of increased kidney weights and possible carcinogenicity in a subchronic oral rat study. The Department of Environmental Protection (New Jersey DEP) has received some reports of taste and odor problems at wells with MTBE in concentrations below 70 µg/L (New Jersey DEP, 2003).

New Jersey has been collecting data on MTBE occurrence in public water supplies since 1997. In the first year of sampling, MTBE was found in 15 percent of community water systems and 16 percent of non-transient non-community water systems. In 400 community water systems, the highest concentration was 8.4 µg/L. In 397 non-transient non-community water systems, two had concentrations exceeding 20 µg/L, and only one exceeded the MCL of 70 µg/L. The State was only able to find obvious sources of contamination for about two thirds of the contaminated PWSs (New Jersey DEP, 2003).

New Jersey stores large amounts of fuel in USTs. New Jersey DEP estimates that the State's 34,000 gasoline-containing USTs have an annual throughput of about 1.7 billion gallons of gasoline, including about 187 million gallons of MTBE (New Jersey DEP, 2003). New Jersey has confirmed reports of 9,383 leaking storage tanks, of which 5,558 have been cleaned up (USEPA, 2005f). The State's Bureau of Underground Storage Tanks (BUST) reported that 80 percent of the 2,400 LUST sites with ground water cleanups had MTBE concentrations above 70 µg/L in ground water (New Jersey DEP, 2003).

The New Jersey District Office of the USGS has been gathering data on MTBE detections in New Jersey surface and ground water through several projects. A compiled summary of all New Jersey data from the Ambient Ground Water Monitoring Network as well as several special studies conducted in the State by USGS is presented in Exhibit 14-20 (NESCAUM, 1999a). Note that the data are aggregated from several studies, and in some cases multiple detections are reported from a single site. According to NESCAUM (1999a), USGS researchers concluded that atmospheric deposition could account for the relatively high frequency of low-level MTBE contamination in New Jersey, though they did not rule out migration of MTBE from distant gasoline release sites. In its own summary of findings and recommendations for the region, NESCAUM (1999b) concludes that low-level MTBE contamination in the Northeast is probably attributable to a combination of atmospheric deposition, small surface spills, and storm water runoff.

Exhibit 14-20: Detection of MTBE in Ground and Surface Waters in New Jersey

Year	No. Samples	No. Detects	Median (µg/L)	Max (µg/L)	Percent detects
Ground Water					
1998	102	35	0.19	4.1	34%
1997	183	55	0.67	30.2	30%
1996	89	34	0.21	43.8	38%
1995	23	5	0.3	2.3	22%
1994	19	3	0.5	2.1	16%
Surface Water					
1998	161	112	1.09	29	70%
1997	98	94	0.52	16	96%
1996	107	80	0.41	4.9	75%
Total					
	782	418			53%

Source: USGS data, presented in NESCAUM, 1999a
Minimum detection limits ranged from 0.5 µg/L to 0.1 µg/L.
Percent detects calculated by dividing the number of detects by the number of samples.

New York

Parts of New York have participated in both the Federal RFG program and the wintertime Oxyfuel program. Both the New York City metro area and the Syracuse area were required to use oxygenated fuel starting in 1992 as part of the Oxyfuel program. Syracuse was released from the requirement in 1993 and New York City in 2000 (USEPA, 2001b). As of February 2004, thirteen counties were participating in the Federal RFG program (USEPA, 2005b).

New York did not require testing for MTBE until July 1998. Now, the New York State Department of Health requires MTBE information from ground water suppliers serving over 10,000 people. Additionally, New York State's Wadsworth Laboratories performs compliance monitoring for small PWSs. Sampling at Wadsworth is not representative of the entire State, but results of PWS samples from 1996 to 1998 are presented in Exhibit 14-21 (NESCAUM, 1999a).

Exhibit 14-21: MTBE in Public Water Systems Samples analyzed by Wadsworth Laboratory in New York

Year	No. Samples	No. Detects	Percent Detects	No. Detects > 10 µg/L	Maximum (µg/L)
1996	282	5	1.8%	5	66
1997	404	17	4.2%	1	17
1998	381	16	4.2%	3	33

Note: The minimum detection limit was 10 µg/L in 1996, either 1 or 10 µg/L in 1997, and 1 µg/L in 1998. The data included multiple samples from some wells.
Source: NESCAUM, 1999a

One study provides an idea of contamination in private wells in New York. Lince *et al.* (2001) sampled 74 private wells near 21 gasoline stations in New York State, plus 21 control wells. Twenty-eight percent of the 74 study wells had MTBE in concentrations at or above the practical quantitation limit of 1 µg/L. The mean concentration was 12 µg/L, and the highest concentration was 61 µg/L. Seven percent of wells had concentrations between 20 and 49 µg/L, and 3 percent had concentrations of 50 µg/L or higher. Five percent of the control wells (1 well) exceeded 1 µg/L. Detections greater than 1 µg/L were more frequent in wells near stations selling reformulated gasoline (38 percent) than near stations selling conventional gasoline (20 percent) (Lince *et al.*, 2001).

In May of 2000, New York State adopted a complete ban of MTBE. The ban became effective January 1, 2004 (USEPA, 2004c).

Washington

Of the three urban areas initially regulated under the wintertime Oxyfuel program, the Seattle and Vancouver vicinities gained exemption from the program in 1996. The third area, Spokane, uses ethanol to meet its oxygenated fuel requirement (USEPA, 2001b). No part of Washington has ever participated in the RFG program (USEPA, 2005b). Washington's Department of Ecology (Washington DOE) has documented the use of MTBE as a fuel oxygenate in the State on only one occasion (Washington DOE, 2000).

A 2000, Washington DOE conducted a study of MTBE contamination in ground water. As of May 2000, Washington had 6,000 regulated LUST sites, of which 1,900 (32 percent) had reportedly impacted ground water. Washington DOE chose 70 geographically representative sites from among the 1,900, and took one monitoring well sample from each. DOE found that 26 sites (42 percent) had detections at or above 1 µg/L and 24 percent had concentrations above 20 µg/L. The highest concentration was 7,150 µg/L. The median concentration of all samples at or exceeding 1 µg/L was 13 µg/L. A measured occurrence rate of 42 percent suggests that over 800 LUST sites might be point sources responsible for MTBE contamination of ground water in the State. However, Washington DOE notes that due to limitations of the study (e.g., sampling of only well per site, irregular quantitation limits, 80% of releases occurred five or more years previously, location of monitoring wells), the actual number might be much higher (Washington DOE, 2000).

The ground water clean-up level established by the State of Washington for MTBE is 20 µg/L (NEIWPCC, 2003). In May of 2001, the State legislature approved a partial ban of MTBE starting January 1, 2004. MTBE may not intentionally be added to fuel or knowingly mixed in gasoline to concentrations above 0.6 percent by volume (USEPA, 2004c).

14.3.6 State MTBE Regulations

At least 25 States have instituted partial or complete bans on MTBE. See Exhibit 14-22 for a list of such States and their phase-out dates.

Exhibit 14-22: State Actions Banning MTBE

State	Effective Date	Extent of MTBE Ban
Arizona	January 1, 2005	0.3% max volume in gasoline
California	December 31, 2003	complete ban in gasoline
Colorado	April 30, 2002	complete ban in gasoline
Connecticut	January 1, 2004	complete ban in gasoline
Illinois	July 24, 2004	0.5% max volume in gasoline
Indiana	July 24, 2004	0.5% max volume in gasoline
Iowa	July 1, 2000	0.5% max volume in gasoline
Kansas	July 1, 2004	0.5% max volume in gasoline
Kentucky	January 1, 2006	0.5% max volume in gasoline
Maine	January 1, 2007	0.5% max volume in gasoline
Michigan	June 1, 2003	complete ban in gasoline
Minnesota	July 2, 2005	complete ban in gasoline (following partial ban in 2000)
Missouri	July 1, 2005	0.5% max volume in gasoline
Montana	January 1, 2006	no more than trace amounts in gasoline
Nebraska	July 13, 2000	1% max volume in gasoline
New Hampshire	January 1, 2007	0.5% max volume in gasoline
New Jersey	January 1, 2009	0.5% max volume in gasoline
New York	January 1, 2004	complete ban in gasoline
North Carolina	January 1, 2008	0.5% max volume in gasoline
Ohio	July 1, 2005	0.5% max volume in gasoline
Rhode Island	June 1, 2007	0.5% max volume in gasoline
South Dakota	July 1, 2001	0.5% max volume in gasoline
Vermont	January 1, 2007	0.5% max volume in gasoline
Washington	January 1, 2004	0.6% max volume in gasoline
Wisconsin	August 1, 2004	0.5% max volume in gasoline

Source: This table is adapted from USEPA, 2004c and McCarthy and Tiemann, 2005. It has been further updated as more recent information has become available in the media. For current information, see individual State regulations.

In 2003, the NEIWPCC conducted a survey of MTBE contamination at LUST sites in the States. All 50 States replied to the survey, though not all States responded to every question. Forty-two States reported that they had action levels, cleanup levels, or drinking water standards for MTBE. Of these, 23 States had soil action levels, 27 had soil cleanup levels, 31 had ground water action levels, and 34 had ground water cleanup levels. Eleven States had established primary drinking water standards for MTBE (See Exhibit 14-23). Four of these States had standards below 20 µg/L, and another four had standards in EPA's health advisory range for organoleptic (taste and odor) effects (20 µg/L to 40 µg/L). The remaining primary drinking water standards were 50 µg/L, 70 µg/L, and 240 µg/L. Seven States had established secondary drinking water standards for MTBE, ranging from 5 µg/L to 400 µg/L. Eleven States had adopted EPA's drinking water advisory level (range of 20-40 µg/L, based on organoleptic effects), and thirteen States had established health advisory levels of their own, ranging from 20 µg/L to 200 µg/L (NEIWPCC, 2003).

Exhibit 14-23 State Primary Drinking Water Standards

State	Primary Drinking Water Standard
California	13 µg/L
Colorado	15 µg/L
Delaware	10 µg/L
Maine	35 µg/L
Mississippi	240 µg/L
Missouri	20 µg/L
New Hampshire	13 µg/L
New Jersey	70 µg/L
New York	50 µg/L
Oregon	20 µg/L
Vermont	40 µg/L

Source: NEIWPCC, 2003

Note: Two additional States, Illinois and Kansas, are listed by NEIWPCC as having primary drinking water standards, but apparently in error, as no numerical value for either standard is provided. Since this table is based on information collected in 2003, it is not guaranteed to be current. For the most up-to-date information and complete details, see State regulations.

In 2000, according to an earlier NEIWPCC survey, 20 of 38 responding States had established action levels for MTBE in soil, 28 had soil clean-up levels, 26 had action levels for MTBE in ground water, and 32 had ground water clean-up levels (NEIWPCC, 2000). In 2003, with all 50 States responding, the numbers were: 23 States with soil action levels, 27 with soil clean-up levels, 31 with ground water action levels, and 34 with ground water clean-up levels (NEIWPCC, 2003).

14.4 References

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. *Toxicological Profile for Automotive Gasoline*. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services. Available on the Internet at: http://www.atsdr.cdc.gov/toxprofiles/tp72.html.

ATSDR. 1996. *Toxicological Profile for Methyl t-Butyl Ether (MTBE)*. Available on the Internet at: http://www.atsdr.cdc.gov/toxprofiles/tp91.html.

ATSDR. 1997. *Toxicological Profile for Benzene*. Available on the Internet at: http://www.atsdr.cdc.gov/toxprofiles/tp3.html.

Alliance for Proper Gasoline Handling. 1999. "New Alliance Launches Consumer "Gas Care" Campaign to Prevent Small Gasoline Spills." Press release dated July 27, 1999. Available on the Internet at: http://www.gas-care.org/Press_release_june13.htm. Accessed March 25, 2005.

American Petroleum Institute (API). 2000. *Strategies for Characterizing Subsurface Releases of Gasoline Containing MTBE*. Publication Number 4699. Available on the Internet at: http://api-ep.api.org/filelibrary/4699c.pdf.

Arizona Department of Environmental Quality (ADEQ). 1999. *Report on Methyl Tertiary Butyl Ether (MTBE)*. Available on the Internet at: http://www.azdeq.gov/environ/ust/download/1001mtbe.pdf.

Baldys, Stanley, III, T.H. Raines, B.L. Mansfield, and J.T. Sandlin. 1998. *Urban stormwater quality, event mean concentrations, and estimates of stormwater pollutant loads, Dallas-Fort Worth area, Texas, 1992–93.* U.S. Geological Survey Water-Resources Investigation Report 98-4158, 51 p.

Bennett, G.M. and W.G. Philip. 1928. *Journal - Chemical Society, London*, pp. 1930-1937. (As cited in HSDB, 2004).

Blue Ribbon Panel on Oxygenates in Gasoline. 1999. Achieving Clean Air and Clean Water. Available on the Internet at: http://www.epa.gov/OMS/consumer/fuels/oxypanel/r99021.pdf.

Borden, R.C., R.A. Daniel, L.E. LeBrun IV, and C.W. Davis. 1997. Intrinsic biodegradation of MTBE and BTEX in a gasoline-contaminated aquifer. *Water Resources Research*. 33(5):1105-1115. (As cited in Chisala *et al.*, 2004).

Bourelle, A. 1998a. MTBE: A Real Concern for South Lake Tahoe. Tahoe Daily Tribune and The Lake Tahoe News. August 28, 1998. Available at: http://www.tahoedailytribune.com.

Bourelle, A. 1998b. STPUD Goes After Major Corporations. Tahoe Daily Tribune and The Lake Tahoe News. November 11, 1998. Available at: http://www.tahoedailytribune.com.

Bradley, P.M., F.H. Chapelle, and J.E. Landmeyer. 2001a. Methyl *t*-butyl ether mineralization in surface-water sediment microcosms under denitrifying conditions. *Applied and Environmental Microbiology.* 67(4):1975-1978.

Bradley, P.M., J.E. Landmeyer, and F.H. Chapelle. 2001b. Widespread Potential for Microbial MTBE Degradation in Surface-Water Sediments. *Environmental Science and Technology.* 35:658-662.

British Environmental Agency. 2002. *A review of the current MTBE usage and occurrence in groundwater in England and Wales.* Environmental Agency, R & D Project Report. No. 97. (As cited in Chisala, 2004).

Bruns, M.A., J.R. Hanson, J. Mefford and K.M. Scow. 2001. Isolate PM1 populations are dominant and novel Methyl-tert-butyl ether-degrading bacteria in compost biofilter enrichments. *Environmental Microbiology*. 3(3): 220-225. (As cited in Zanardini, 2002).

California Department of Health Services (California DHS). 2000. Final Statement of Reasons: Primary Maximum Contaminant Level for Methyl tert-Butyl Ether. Available at www.dhs.ca.gov/ps/ddwem/chemicals/mtbe/4-17-00MTBESOR.PDF.

California DHS. 2002. *Annual Compliance Report for Public Water Systems, Calendar Year 2002*. Available on the Internet at: http://www.dhs.ca.gov/ps/ddwem/publications/2002annualcompliancereport-final.pdf. Link to document from: http://www.dhs.ca.gov/ps/ddwem/publications/pubindex.htm.

California DHS. 2003. *Annual Compliance Report for Public Water Systems, Calendar Year 2003*. Available on the Internet at: http://www.dhs.ca.gov/ps/ddwem/publications/2003ARCFinal.pdf. Link to document from: http://www.dhs.ca.gov/ps/ddwem/publications/pubindex.htm.

California DHS. 2004a. MTBE: regulations, the drinking water treatment and research funds, and monitoring results. Includes link to downloadable occurrence data. Available on the Internet at: http://www.dhs.ca.gov/ps/ddwem/chemicals/MTBE/mtbeindex.htm. Accessed December 20, 2004. Last updated December 8, 2004.

California DHS. 2004b. *Annual Compliance Report for Public Water Systems, Calendar Year 2004*. Available on the Internet at: http://www.dhs.ca.gov/ps/ddwem/publications/AnnualComplianceReport2004.pdf. Link to document from: http://www.dhs.ca.gov/ps/ddwem/publications/pubindex.htm.

California DHS. 2005. MTBEforweb. Excel spreadsheet. Available on the Internet at: http://www.dhs.ca.gov/ps/ddwem/chemicals/MTBE/mtbeforweb.XLS. Explanation and link to spreadsheet from: http://www.dhs.ca.gov/ps/ddwem/chemicals/MTBE/mtbeindex.htm. Accessed March 27, 2006. Last updated October 18, 2005.

California Environmental Protection Agency (CAEPA). 1994. *California RFG Forum: California Phase 2 Reformulated Gasoline News*. No. 1. December. Air Resources Board. Available on the Internet at: http://www.arb.ca.gov/fuels/gasoline/pub/forum1.pdf.

Chisala, B.N., N.G. Tait, and D.N. Lerner. 2004. Evaluating the risk of methyl tertiary-butyl ether (MTBE) to urban groundwater. Hydrology: Science and Practice for the 21st Century. Volume II: 243-249.

Church, C.D., P.J. Tratnyek, J.F. Pankow, J.E. Landmeyer, A.L. Baehr, M.A. Thomas, and M. Schirmer. 1999. Effects of environmental conditions on MTBE degradation in model column aquifers. In: Proceedings of the Technical Meeting of USGS Toxic Substances Hydrology Program, Vol 3 (pp 93-101). Charleston, SC. (As cited in Deeb, 2000).

Connecticut Department of Environmental Protection (Connecticut DEP). 2000. Report to the Joint Standing Committees on Public Health and the Environment Connecticut General Assembly. Hartford, CT: State of Connecticut. Available on the Internet at: http://www.dep.state.ct.us/air2/mtbe/reports/mtbe00.pdf.

Connecticut DEP. 2004. MTBE - Methyl Tertiary Butyl Ether. Webpage. http://www.dep.state.ct.us/air2/mtbe/index.htm. Accessed May 13, 2005. Last copyrighted 2004.

Cook, G.E., L. R. Davis, and C. Tulloch. 2002. *Evaluation of MtBE Occurrence at Operating Gasoline UST Facilities in Santa Clara County, California: Preliminary Findings*. Santa Clara Valley Water District report. Available on the Internet at: http://www.valleywater.org/Water/Water_Quality/Protecting_your_water/_Lustop/_Publications_and_Documents/_PDFs_Etc/Eval_of_MTBE_Occurrence.pdf.

Crofton, G. 2004. Tahoe Water Supply on the Rebound. Tahoe Daily Tribune and The Lake Tahoe News. July 27, 2004. Available at: http://www.tahoedailytribune.com.

Dahlen, P., E. Henry, M. Matsumura, and P.C. Johnson. 2003. *Impacts to Groundwater Resources in Arizona from Leaking Underground Storage Tanks (LUSTS)*. Arizona State University, Arizona Department of Environmental Quality. Available on the Internet at: http://www.eas.asu.edu/civil/Environmental/Main_Report/Impacts%20to%20GW%20Resources%20in%20AZ%20from%20Leaking%20USTs.pdf.

Dakhel, N, G. Pasteris, D. Werner, and P. Hohener. 2003. Small-Volume Releases of Gasoline in the Vadose Zone: Impact of the Additives MTBE and Ethanol on Groundwater Quality. *Environmental Science & Technology*. 37:2127-2133.

Day, M.J., R.F. Reinke, and J.A.M. Thomson. 2001. Fate and Transport of Fuel Components Below Slightly Leaking Underground Storage Tanks. *Environmental Forensics.* 2:21-28.

Deeb, R.A, K.M. Scow, and L. Alvarez-Cohen. 2000. Aerobic MTBE biodegradation: an examination of past studies, current challenges and future research directions. *Biodegradation.* 11:171-186.

Deeb, R.A., K. Chu, T. Shih, S. Linder, M. Suffett, M.C. Kavanaugh, and L. Alvarez-Cohen. 2003. MTBE and other oxygenates: environmental sources, analysis, occurrence, and treatment. *Environmental Engineering Science*. 20(5):433-447.

Delzer, G.C. and T. Ivahnenko. 2003a. *A Review of Literature for Methyl tert-Butyl Ether (MTBE) in Sources of Drinking Water in the United States.* U.S. Geological Survey Open-File Report 01-322, 16 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr01_322.pdf.

Delzer, G.C. and T. Ivahnenko. 2003b. *Occurrence and Temporal Variability of Methyl tert-Butyl Ether (MTBE) and Other Volatile Organic Compounds in Select Sources of Drinking Water: Results of the Focused Survey.* U.S. Geological Survey Water-Resources Investigations Report 02-4084, 65 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4084.pdf.

Delzer, G.C., J.S. Zogorski, T.J. Lopes, and R.L. Bosshart. 1996. *Occurrence of the Gasoline Oxygenate MTBE and BTEX Compounds in Urban Stormwater in the United States, 1991-95.* USGS Water Resources Investigations Report 96-4145. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir96.4145/wrir.doc.html.

Dodds, R.S. 2004. *MTBE Quarterly Report (Jan-Mar 2004).* April 6. Available on the Internet at: http://www.waterboards.ca.gov/lahontan/MTBE/MTBE_Rpt_Jan_Mar_2004.pdf.

Environmental Working Group (EWG). 2005. Environmental Working Group. Like Oil and Water. Available on the Internet at: www.ewg.org/reports/oilandwater. Accessed August 29, 2006.

Fayolle, F., J.P. Vandecasteele, and F. Monot. 2001. Microbial degradation and fate in the environment of methyl tert-butyl ether and related fuel oxygenates. *Applied Microbiology and Biotechnology.* 56:339-349.

Feliciano, D.V. 1984. *Leaking underground storage tanks: A potential environmental problem.* Washington, DC: Library of Congress, Congressional Research Service, January 11. Report no.
84-508ENR. (As cited in ATSDR, 1995).

Finneran, K.T. and D.R. Lovley. 2001. Anaerobic Degradation of Methyl tert-Butyl Ether (MTBE) and tert-Butyl Alcohol (TBA) [in Aquifer Sediments]. *Environmental Science and Technology.* 35:1785-1790.

Fiorenza, S. and H.S. Rifai. 2003. Review of MTBE Biodegradation and Bioremediation. *Bioremediation Journal.* 7(1):1-35.

Florida Department of Environmental Protection (Florida DEP). 2004a. MTBE in Florida Groundwater Resources. Available on the Internet at: http://www.dep.state.fl.us/water/drinkingwater/docs/MtBE_in_FL_GW.pdf.

Florida DEP. 2004b. MtBE (Methyl Tertiary-Butyl Ether) Occurrence in Florida Drinking Water Systems. Available on the Internet at: http://www.dep.state.fl.us/water/drinkingwater/mtbe.htm. Accessed December 20, 2004. Last updated June 8, 2005.

Florida DEP. 2004c. MTBE Sampling Results. Available for download at:: http://www.dep.state.fl.us/water/drinkingwater/mtbesamp.htm. Accessed December 20, 2004. Last updated March 6, 2004.

Garnier, P., R. Auria, C. Auger, and S. Revah. 1999. Cometabolic biodegradation of methyl t-butyl ether by *Pseudomonas aeruginosa* grown on pentane. *Applied Microbiology and Biotechnology.* 51: 498-503. (As cited in Stocking *et al.*, 2000).

Garrett, P., M. Moreau, and J.D. Lowry. 1986. MTBE as a Ground Water Contaminant. Proceedings of the 1986 Conference on Petroleum and Organic Chemicals in Ground Water, National Water Well Association and American Petroleum Institute, Houston, TX, p. 227. (As cited in NESCAUM, 1999a).

Gilbert, C.E. and E.J. Calabrese. 1992. Developing a standard for methyl butyl ether in drinking water. In: Gilbert, C.E, and Calabrese, E.J., eds. *Regulating Drinking Water Quality*. Lewis Publishers, 23l-252. (As cited in ATSDR, 1996).

Grady, S.J. 2003. *A National Survey of Methyl tert-Butyl Ether and Other Volatile Organic Compounds in Drinking-Water Sources: Results of the Random Survey.* U.S. Geological Survey Water-Resources Investigations Report 02-4079, 85 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4079.pdf.

Grady, S.J. and G.D. Casey. 2001. *Occurrence and Distribution of Methyl Tert-Butyl Ether and Other Volatile Organic Compounds in Drinking Water in the Northeast and Mid-Atlantic Regions of the United States, 1993-98.* U.S. Geological Survey Water-Resources Investigations Report 00-4228, 128 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir00.4228.pdf .

Gullick, R. and M.W. LeChevallier. 2000. Occurrence of MTBE in Drinking Water Sources. *Journal AWWA.* January. 92:100-113.

Happel, A.M., E.H. Beckenbach, and R.U. Halden. 1998. An evaluation of MTBE impacts to California groundwater resources. Lawrence Livermore National Laboratory Report, UCRL-AR-130897.

Happel, A.M., E.H. Beckenbach, and R.U. Halden. 1999. MTBE Impacts to California Groundwater Resources. Presentation to the USEPA Blue Ribbon Panel, March 25, 1999. Draft meeting minutes are available on the Internet at: http://www.epa.gov/otaq/consumer/fuels/oxypanel/sacdraft.pdf.

Hardison, L.K., S.S. Curry, L.M. Ciuffetti, and M.R. Hyman. 1997. Metabolism of Diethyl Ether and Cometabolism of Methyl tert-Butyl Ether by a Filamentous Fungus, a *Graphium* sp. *Applied and Environmental Microbiology*. 63(8):3059-3067.

Hatten, G. 2000. "MTBE Treatment Systems in Kansas." In: Association of State and Territorial Solid Waste Management Officials (ASTSWMO) MTBE and Fuel Oxygenates Workgroup Newsletter. 3(1). March.

Hatten, G. and G. Blackburn. 1999. "Findings of Kansas MTBE Investigation." In: Kansas Environmental News - April, 1999. Available online at http://www.kdhe.state.ks.us/sbcs/download/ken9904.html#mtbe. Accessed December 8, 2004.

Hawaii Department of Health. 1998. Policy Statement: Requirement for Sampling and Analysis of Methyl tertiary-Butyl Ether in Soil and Groundwater at Underground Storage Tank Closure and Release Investigation Sites. Honolulu: State of Hawaii. Available on the Internet at: http://www.hawaii.gov/health/environmental/waste/ust/pdf/mtbe.pdf.

Hawaii DOH. 2004. Trace Amounts of Chemical Found in Wahiawa Well. News Release, November 23. Available on the Internet at: http://state.hi.us/health/about/pr/2004/2004/04-86sdwb.mtbe.pdf.

Hazardous Substances Data Bank (HSDB). 2004. Search for MTBE. Available on the Internet through TOXNET, sponsored by the National Institute of Health's National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB. Accessed October 25, 2004.

Hine J. and P.K. Mookerjee. 1975. The intrinsic hydrophilic character of organic compounds. correlations in terms of structural contributions. *Journal of Organic Chemistry*. 40(3):292-296. (As cited in ATSDR, 1996).

Howard, P.H., R.S. Boethling, W.F. Jarvis, W.M. Meylan, and E.M. Michalenko. 1991. Handbook of Environmental Degradation Rates. Lewis, Chelsea, MI. (As cited in Squillace *et al.*, 1997).

Iowa Department of Natural Resources (IDNR). 2000. Methyl tertiary-Butyl Ether (MTBE) Occurrence in Iowa: A report for the 2000 session of the Seventy-Eighth General Assembly. Des Moines, IA: IDNR. Available on the Internet at: http://www.iowadnr.com/land/ust/technicalresources/mtbe/documents/mtbereport.pdf.

Ivahnenko, T., S.J. Grady, and G.C. Delzer. 2001. *Design of a National Survey of Methyl tert-Butyl Ether and Other Volatile Organic Compounds in Drinking-Water Sources.* U.S. Geological Survey Open-File Report 01-271, 42 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr01_271.pdf.

Javanmardian, M. and H.A. Glasser. 1997. In-situ biodegradation of MTBE using biosparging. Preprints of Extended Abstracts. *American Chemical Society*. 37:424-426. (As cited in Deeb *et al.*, 2000).

Johnson, R, J. Pankow, D. Bender, C. Price, and J. Zogorski. 2000. MTBE: To What Extent Will Past Releases Contaminate Community Water Supply Wells? *Environmental Science and Technology.* 210A-217A.

Kansas Department of Health and Environment (KDHE). 2000. KDHE Responds to Concerns Over MTBE. Press release, January 21. Available on the Internet at: http://www.kdheks.gov/news/web_archives/2000/00-014.htm.

Landmeyer, J.E., F.H. Chapelle, H.H. Herlong, and P.M. Bradley. 2001. Methyl tert-Butyl Ether Biodegradation by Indigenous Aquifer Mircroorganisms under Natural and Artificial Oxic Conditions. *Environmental Science and Technology*. 35:1118-1126.

Levine-Fricke. 1999. *Summary Report: Santa Clara Valley Water District Groundwater Vulnerability Pilot Study: Investigation of MTBE Occurrence Associated with Operating UST Systems*. Volume 1 of 3. July 22, 1999. Available on the Internet at: http://www.valleywater.org/Water/Technical_Information/Technical_Reports/_Reports/TextTables.pdf.

Lide, D.R.. 1994. *CRC Handbook of Chemistry and Physics*. 74th edition, CRC Press Boca Raton 3-146. (As cited in ATSDR, 1996).

Lidderdale, T. and A. Bohn. 1999. Demand and Price Outlook for Phase 2 Reformulated Gasoline, 2000. U.S. Energy Information Administration. First published April 1999, revised August 1999. Available on the Internet at: http://www.eia.doe.gov/emeu/steo/pub/special/rfg4.html.

Lidderdale, T. 2003. Motor Gasoline Outlook and State MTBE Bans. U.S. Energy Information Administration. File last modified April 6, 2003. Available on the Internet at: http://www.eia.doe.gov/emeu/steo/pub/special/mtbeban.html.

Lince, D.P., L.R. Wilson, G.A. Carlson, and A. Bucciferro. 2001. Effects of gasoline formulation on Methyl tert-Butyl Ether (MTBE) contamination in private wells near gasoline stations. *Environmental Science & Technology*. 35:1050-1053.

Lopes, T.J., K.D. Fossum, J.V. Phillips, and J.E. Monical. 1995. *Statistical summary of selected physical, chemical, and microbial characteristics, and estimates of constituent loads in urban stormwater, Maricopa County, Arizona.* U.S. Geological Survey Water-Resources Investigations Report 94-4240.

Lopes, T.J. and S.G. Dionne. 1998. *A Review of Semivolatile and Volatile Organic Compounds in Highway Runoff and Urban Stormwater.* U.S. Geological Survey Open-File Report 98-409, 67 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/ofr/ofr98-409.pdf.

Los Angeles Regional Water Quality Control Board (LARWQCB) and United States Environmental Protection Agency (USEPA) Region 9. 1998. MtBE Project Fact Sheet. Available on the Internet at: http://www.epa.gov/region09/cross_pr/mtbe/charnock/techdocs/july_98_fs.pdf. More recent information on the Charnock site and link to document at: http://www.epa.gov/region09/cross_pr/mtbe/charnock/discovery.html. Accessed December 7, 2004.

Lynn, G. 2004. Tracking Troubling Vapor Releases in New Hampshire. *LustLine Bulletin*. Vol. 47. June. Available on the Internet at: http://www.neiwpcc.org/PDF_Docs/l_line47.pdf.

Mackay D. and P.J. Leinonen. 1975. Rate of evaporation of low-solubility contaminants from water bodies to atmosphere. *Environmental Science and Technology*. 9:1178-1180. (As cited in ATSDR, 1997).

Mace, R.E., S. Fisher, D.M. Welch, and S.P. Parra. 1997. *Extent, Mass, and Duration of Hydrocarbon Plumes from Leaking Petroleum Storage Tank Sites in Texas.* Bureau of Economic Geology, The University of Austin, Texas. (As cited in Stocking *et al.*, 2000).

Maine. 1998. The Presence of MTBE and Other Gasoline Compounds in Maine's Drinking Water: A Preliminary Report. October 13. Maine Bureau of Health Department of Human Services, Maine Bureau of Waste Management & Remediation Department of Environmental Protection, and Maine Geological Survey Department of Conservation. Available on the Internet at: http://www.maine.gov/dhhs/ehu/wells/MTBE.PDF.

Maine Department of Environmental Protection (Maine DEP). 2005. 2004 Maine Fuels Report. Bureau of Air Quality. Available on the Internet at: http://mainegov-images.informe.org/dep/air/mobile/2004fuelsrpt.pdf.

Maryland Department of the Environment (MDE). 2002. Methyl Tertiary-Butyl Ether (MTBE) Recent Developments as of May 2002. Available on the Internet at: http://www.mde.state.md.us/Programs/LandPrograms/Oil_Control/mtbe_update/index.asp. Accessed December 20, 2004.

Maryland Task Force. 2001. Final Report of the Task Force on the Environmental Effects of Methyl tert-Butyl Ether (MTBE), December, 2001. Available on the Internet at: http://www.mde.state.md.us/assets/document/waste/mtbe_finalreport.pdf.

McCarthy, J.E. and M. Tiemann. 2005. *MTBE in Gasoline: Clean Air and Drinking Water Issues.* CRS Report for Congress. Updated August 17, 2005. Available on the Internet at: http://www.cnie.org/nle/crsreports/05aug/RL32787.pdf.

Mello, M. 2001. "Tainted water worries R.I. town." Associated Press, November 13, 2001. Available online at: http://www.bbiethanol.com/news/view.cgi?article=374. Accessed December 9, 2004.

Merck. 1989. *Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals*. 11th ed. Budavari S, ed. Rahway NJ: Merck & Co., Inc. (As cited in ATSDR, 1996 and ATSDR, 1997).

Michigan Department of Agriculture (MDA). 2002. MTBE Report, May 31, 2002. Motor Fuels Quality Program. Available on the Internet at: www.deq.state.mi.us/documents/deq-std-MTBE1.doc. Accessed December 17, 2004. Last updated June 12, 2002.

Michigan Department of Environmental Quality (MDEQ). 2000. MTBE (methyl tertiary butyl ether) fact sheet. Available on the Internet at: http://www.deq.state.mi.us/documents/deq-std-MTBE.pdf. (As cited in Delzer and Ivahnenko, 2003a).

Missouri Department of Natural Resources (MDNR). 2004a. MTBE Summary of Facts. Fact Sheet, February, 2004. Available on the Internet at: http://www.dnr.mo.gov/pubs/pub2080.pdf.

Missouri Department of Natural Resources (MDNR). 2004b. Currently Active Sites where MTBE has been detected at a public or private drinking water well. Updated Table from MDNR, 2004a.

Mo, K., C.O. Lora, A.E. Wanken, M. Javanmardian, X. Yang, and C.F. Kulpa. 1997. Biodegradation of methyl *t-butyl* ether by pure bacterial cultures. *Applied Microbiology and Biotechnology.* 47(1):69-72. (As cited in Zanardini *et al.*, 2002).

Moeri, E.N., M.C. Salvador, and R. Coelho. 2001. Natural attenuation of MTBE as part of an integrated remediation design. In: *Bioremediation of MTBE, Alcohols and Ethers*. V.S. Magar, J.T. Gibbs, K.T. O'Reilly, M.R. Hyman, and A. Leeson (eds.) *Bioremediation series.* 6:27-34. (As cited in Chisala *et al.*, 2004).

Moran, M.J., W.W. Lapham, B.L. Rowe, and J.S. Zogorski. 2002. *Occurrence and Status of Volatile Organic Compounds in Ground Water from Rural, Untreated, Self-Supplied Domestic Wells in the United States, 1986-1999*. U.S. Geological Survey Water-Resources Investigations Report 02-4085, 51 p. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/wrir/wrir02_4085.pdf.

Moyer, E.E. and T.P Kostecki. 2003. *MTBE Remediation Handbook*. Amherst Scientific Publishers. Massachusetts. (As cited in Chisala *et al.*, 2004).

National Conference of State Legislators (NCSL). 2000. Issue Brief: Methyl Tertiary Butyl Ether (MTBE). Prepared for the NCSL Clean Air Working Group and AFI Environment Committee. NCSL Annual Meeting, July 2000. Available on the Internet at: http://www.ncsl.org/statefed/0ibmtbe.htm. Accessed May 13, 2005.

Naval Facilities Engineering Command (NFESC). 2000. TechData Sheet: Methyl Tertiary Butyl Ether (MTBE) Bioremediation. TDS-2081-ENV. Available on the Internet at: http://enviro.nfesc.navy.mil/erb/erb_a/restoration/fcs_area/mtbe/tds-2081-mtbe.pdf.

Northeast States for Coordinated Air Use Management (NESCAUM). 1999a. Attachment II: MTBE in Ground and Surface Waters of the NESCAUM Region. August. Available online at:

http://72.14.203.104/search?q=cache:gsDIU362fHwJ:www.nescaum.org/pdf/MTBE_PH2/Ph2water.pdf+Attachment+II:+MTBE+in+Ground+and+Surface+Waters+of+the+NESCAUM+Region&hl=en&gl=us&ct=clnk&cd=1.

NESCAUM. 1999b. RFG/MTBE: Findings and Recommendations. August.

New England Interstate Water Pollution Control Commission (NEIWPCC). 2000. 2000 NEIWPCC Survey of MtBE at LUST Sites. Available on the Internet at: http://www.neiwpcc.org/mtbe.htm.

NEIWPCC. 2003. 2003 NEIWPCC Survey of Oxygenates at LUST Sites. Available on the Internet at: http://www.neiwpcc.org/mtbe.htm.

Nevada Department of Environmental Protection (NDEP). 1998. NDEP Oxygenated Fuel Corrective Action Guidance, October 1998. Available on the Internet at: http://ndep.nv.gov/bca/mtbe_doc.pdf.

New Hampshire. 2005. "Governor Lynch Signs Legislation Banning Gasoline with MtBE after January 1, 2007." Press Release. May 12, 2005. Available on the Internet at: http://www.nh.gov/governor/news/051205MtBE.htm. Accessed July 27, 2005.

New Hampshire Department of Environmental Services (NHDES). 2000a. Study of Reformulated Gasoline (RFG) Distributed Outside of New Hampshire's Four County Nonattainment Area. December 1, 2000. Available on the Internet at: http://www.des.state.nh.us/ard/rfgstudy.pdf.

NHDES. 2000b. State adopts tighter water quality standard for the gasoline additive MtBE–new health standard will be the most protective in the nation. NHDES Environmental Bulletin, May 9, 2000.

NHDES. 2002. Revision of the New Hampshire State Implementation Plan for Adoption of Measures to Opt-Out of RFG Program. Available on the Internet at: http://www.des.state.nh.us/ard/pdf/103002_Final_OFRFG_SIP.pdf.

New Jersey Department of Environmental Protection (New Jersey DEP). 2003. MTBE in New Jersey's Environment. Available on the Internet at: http://www.state.nj.us/dep/dsr/mtbe/mtbe-report.htm.

Nielson, M. G. and J. M. Peckenham. 2000. *Methyl tert-Butyl Ether (MTBE) in Ground Water, Air, and Precipitation at North Windham, Maine*. Water-Resources Investigations Report 00-4048, 28 p. U.S. Geological Survey: Augusta, Maine. Available online at: http://me.water.usgs.gov/wrir00-4048.pdf.

Petroleum Storage Tank Insurance Fund of Missouri (PSTIF). 2000. Report on Inter-Agency Meetings on MTBE. Available on the Internet at: http://www.pstif.org/apps/mtbe_report.pdf.

Primedia Business Magazines and Media Inc. (Primedia). 2002. Water Supply: California City Begins MTBE Removal Efforts. September 1. Available on the Internet at: http://www.americancityandcounty.com/mag/government_california_city_begins. Accessed May 25, 2005.

Rhode Island (RI) Department of Health. 2001. "New test results of the water in the Pascoag Utility District water system." Media Release, November 21, 2001. Available online at http://www.health.ri.gov/media/011121b.php. Accessed December 9, 2004.

RI Department of Health. 2002. "Results of tests by the Department of Health meet drinking water standards." Media Release, January 19, 2002. Available online at http://www.health.ri.gov/media/020119a.php. Accessed December 9, 2004.

Rice, D.W., R.D. Grose, J.C. Michaelsen, B.P. Dooher, D.H. MacQueen, S.J. Cullen, W.F. Kastenberg, L.E. Everett and M.A. Marino. 1995. California Leaking Underground Fuel Tank (LUFT) Historical Case Analyses. Lawrence Livermore National Laboratory, UCRL-AR-122207. (As cited in Stocking *et al.*, 2000).

Salanitro, J.P., P.B. Dorn, and T.E. Vipond. 1998. Assessment of the acute toxicity of crude oils in soils using earthworms. *Chemosphere*. 37(5):845-860. (As cited in Zanardini, 2002).

Salanitro, J., G. Spinnler, P. Maner, H. Wisniewski, and P. Johnson. 1999. Potential for MTBE bioremediation - in situ inoculation of specialized cultures. In"Proceedings of the API/NGWA Conference on Petroleum Hydrocarbons and Organic Chemicals in Ground Water: Prevention, Detection and Remediation Conference, 17-20 November, Houston, TX. (As cited in Deeb, 2000).

Sabine River Authority of Texas (SRA). 2000. Summary report of MtBE contamination in Lake Tawakoni from gasoline pipeline rupture. SRA00230. Available online at http://www.sra.dst.tx.us/srwmp/special_reports/lake_tawakoni_gasoline_spill/. Accessed December 9, 2004.

Schirmer, M., B.J. Butler, J.F. Barker, C.D. Church, and K. Schirmer. 1999. Evaluation of Biodegradation and Dispersion as Natural Attenuation Processes of MTBE and Benzene at the Borden Site. *Physics and Chemistry of the Earth (B).* 24(6):557-560. (As cited in Chisala *et al.*, 2004).

Schirmer, M., J.B. Butler, D.C. Church, and F.J. Barker, and N. Nadarajah. 2003. Laboratory evidence of MTBE biodegradation in Borden aquifer materials. *Journal of Contaminant Hydrology.* 60:229-249. (As cited in Chisala *et al.*, 2004).

Seymour, C. 2000. Gas Spill Threat Diminished, Local Water Supplies Safe. The Eagle-Tribune, January 30, 2000. Available online at: http://www.eagletribune.com/framesets/searcharch.htm (Select "2000," "January 30," "Local News"). Accessed December 10, 2004.

Shively, A. 2004. Vermont Vapor Stories: The Evolution of Vapor-Release Assessment Techniques. *LustLine Bulletin.* Vol. 48. November. Available on the Internet at: http://www.neiwpcc.org/PDF_Docs/lustline_48.pdf.

Speth, T.F. and R.J. Miltner. 1990. Adsorption Capacity of GAC for Synthetic Organics. *Journal of American Water Works Association*. 82(2):72-75. (As cited in Speth *et al.*, 2001).

Speth, T.F., M.L. Magnuson, C.A. Kelty, and C.J. Parrett. 2001. Treatment Studies of CCL Contaminants. In: *Proceedings, AWWA Water Quality Technology Conference*. Nashville, TN. November 11-15, 2001.

Squillace, P.J., J.F. Pankow, N.E. Korte, and J.S. Zogorski. 1996. *Environmental Behavior and Fate of Methyl tert Butyl Ether (MTBE).* U.S. Geological Survey Fact Sheet, FS-203-96, 6 pp (Revised February, 1998). Available on the Internet at: http://ca.water.usgs.gov/mtbe/fs20396/index.html. Accessed September 9, 2002.

Squillace, P.J., J.F. Pankow, N.E. Korte, and J.S. Zogorski. 1997. Review of the Environmental Behavior and Fate of Methyl tert-Butyl Ether. *Environmental Toxicology and Chemistry.* 16(9):1836-1844.

Squillace, P.J., M.J. Moran, W.W. Lapham, C.V. Price, R.M. Clawges, and J.S. Zogorski. 1999. Volatile Organic Compounds in Untreated Ambient Groundwater of the United States, 1985-1995. *Environmental Science and Technology*. 33(23):4176-4187. Available on the Internet at: http://sd.water.usgs.gov/nawqa/pubs/journal/EST.voc.squillace.pdf.

Steffan, R.J., K. McClay, S. Vainberg, C.W. Condee, and D. Zhang. 1997. Biodegradation of the Gasoline Oxygenates Methyl tert-Butyl Ether, Ethyl tert-Butyl Ether, and tert-Amyl Methyl Ether by Propane-Oxidizing Bacteria. *Applied and Environmental Microbiology.* 63(11):4216-4222. (As cited in Zanardini, 2002).

Stocking, A.J., R.A. Deeb, A.E. Flores, W. Stringfellow, J.Talley, R. Brownell, and M.C. Kavanaugh. 2000. Bioremediation of MTBE: a review from a practical perspective. *Biodegradation.* 11:187-201.

Sullivan, E.O. 1998. United States Senate Committee on Environment and Public Works: Testimony of Mr. Edward O. Sullivan, Commissioner Department of Environmental Protection [Maine]. September 16. Available on the Internet at: http://epw.senate.gov/105th/sul_9-16.htm.

Tulloch, C. 2000. *An Evaluation of MTBE Occurrence at Fuel Leak Sites with Operating Gasoline USTs*. Santa Clara Valley Water District report. Available on the Internet at: http://www.valleywater.org/Water/Water_Quality/Protecting_your_water/_Lustop/_Publications_and_Documents/_PDFs_Etc/UST_MTBE_Study_Final.pdf.

United States Department of Energy (USDOE). 2000. MTBE, Oxygenates, and Motor Gasoline. Available on the Internet at: http://www.eia.doe.gov/emeu/steo/pub/special/mtbe.html. Last modified March 6, 2000.

USDOE. 2003. Status and Impacts of MTBE Bans. Available on the Internet at: http://www.eia.doe.gov/oiaf/servicerpt/mtbeban/index.html. Includes hyperlinks to tables–domestic consumption data is in Table 2, http://www.eia.doe.gov/oiaf/servicerpt/mtbeban/table2.html. Last modified March 27, 2003.

USDOE. 2005. EIA-819M Monthly Oxygenate Report: Historical. (Consulted individual reports. Reports with data for the last month of each year also contain cumulative data for the whole year.) Available on the Internet at: http://www.eia.doe.gov/oil_gas/petroleum/data_publications/monthly_oxygenate_telephone_report/motr_historical.html. Accessed October 13, 2005.

USDOE. 2006. Annual Energy Outlook 2006, With Projections to 2030. DOE/EIA-0383(2006). February. Available on the Internet at: http://www.eia.doe.gov/oiaf/aeo/pdf/0383(2006).pdf. Link to document and individual chapters from: http://www.eia.doe.gov/oiaf/aeo/index.html.

United States Environmental Protection Agency (USEPA). 1995a. *Toxic chemical inventory reporting Form R and instructions*. EPA Report 745-K-95-051. Washington, DC: Office of Pollution Prevention and Toxics, USEPA. (As cited in ATSDR, 1996).

USEPA. 1995b. Method 502.2 - Volatile Organic Compounds in Water by Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series. (Revision 2.1). In: *Methods for the Determination of Organic Compounds in Drinking Water, Supplement 3*. EPA Report 600-R-95-131.

USEPA. 1997. Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Methyl Tertiary-Butyl Ether (MTBE). EPA Report 822-F-97-009. Washington, DC. December 1997. Available on the Internet at: http://www.epa.gov/waterscience/drinking/mtbe.pdf.

USEPA. 1998a. *Oxygenates in Water: Critical Information and Research Needs*. EPA Report 600-R-98-048.

USEPA. 1998b. *MTBE Fact Sheet #3: Use and Distribution of MTBE and Ethanol, USEPA Office of Underground Storage Tanks*. EPA Report 625-K-98-001. January. Available on the Internet at: http://www.epa.gov/swerust1/mtbe/mtbefs3.pdf.

USEPA. 2000a. Oxygenate-Type Analysis: 1995-2000 Reformulated Gasoline (RFG) Survey Data. Available on the Internet at: http://www.epa.gov/otaq/consumer/fuels/mtbe/oxy-95-00.pdf.

USEPA. 2000b. *Unregulated Contaminant Monitoring Regulation Analytical Methods and Quality Control Manual*. EPA Report 815-R-00-006.

USEPA. 2001a. Summary of Workshop on Biodegradation of MTBE: February 1-3, 2000. Workshop sponsored by EPA and the American Petroleum Institute. EPA Report 625-R-01-

001A. Available on the Internet at: http://www.epa.gov/ORD/NRMRL/pubs/625r01001/625r01001.pdf.

USEPA. 2001b. State Winter Oxygenated Fuel Program Requirements for Attainment or Maintenance of CO NAAQS. Available on the Internet at: http://www.epa.gov/oms/oxygenate.htm. Accessed December 20, 2004.

USEPA. 2002a. *Community Water System Survey 2000. Volume I: Overview*. EPA Report 815-R-02-005A. December 2002. Available on the Internet at: http://www.epa.gov/safewater/consumer/pdf/cwss_2000_volume_i.pdf.

USEPA. 2002b. *Community Water System Survey 2000. Volume II: Detailed Tables and Survey Methodology*. EPA Report 815-R-02-005B. Available on the Internet at: http://www.epa.gov/safewater/consumer/pdf/cwss_2000_volume_ii.pdf.

USEPA. 2003a. Oxygenate Identification. Available on the Internet at: http://www.epa.gov/OUST/oxygenat/oxytable.htm. Accessed October 4, 2004. Last updated March 2003.

USEPA. 2003b. "EPA Announces $1 Million for Pascoag MTBE Cleanup; Funding Boosts EPA Support for Cleanup to $2.4 Million." Available online at http://www.epa.gov/boston/pr/2003/jun/030636.html. Accessed December 9, 2004.

USEPA. 2004a. Stage II Vapor Recovery Systems Issues Paper. Office of Air Quality Planning and Standards, Emissions Monitoring and Analysis Division, Emissions Factors and Policy Applications Group (D243-02). August 12. Available on the Internet at: http://www.epa.gov/ttn/naaqs/ozone/ozonetech/stage2/isspaper081204.pdf.

USEPA. 2004b. State Investigation Reports on MTBE. Available on the Internet at: http://www.epa.gov/oust/mtbe/mtbestat.htm. Last updated June 3, 2004.

USEPA. 2004c. State Actions Banning MTBE (Statewide). EPA Report 420-B-04-009. June 2004. Available on the Internet at: http://www.epa.gov/mtbe/420b04009.pdf.

USEPA. 2005a. Gasoline Fuels [Webpage]. Available on the Internet at: http://www.epa.gov/otaq/gasoline.htm. Accessed March 8, 2005. Last updated February 14, 2005.

USEPA. 2005b. Where You Live: Map of Current RFG Areas [Webpage]. Available on the Internet at: http://www.epa.gov/otaq/rfg/whereyoulive.htm. Accessed March 30, 2006. Last updated September 12, 2005.

USEPA. 2005c. UST Program Facts. Washington, D.C.: Office of Solid Waste and Emergency Response. February 2005. Available on the Internet at: http://www.epa.gov/oust/pubs/ustfacts.pdf.

USEPA. 2005d. Drinking Water Replacement [Webpage]. Available on the Internet at: http://www.epa.gov/region09/cross_pr/mtbe/charnock/drinking.html. Accessed April 29, 2005. Last updated February 16, 2005.

USEPA. 2005e. MTBE Source Investigations [Webpage]. Available on the Internet at: http://www.epa.gov/region09/cross_pr/mtbe/charnock/sourceinvest.html. Accessed April 29, 2005. Last updated February 16, 2005.

USEPA. 2005f. Underground Storage Tanks: Where You Live [Webpage]. Available on the Internet at: http://www.epa.gov/oust/wheruliv.htm. Last updated January 3, 2005.

USEPA. 2006a. IRIS Chemical Assessment Tracking System. Search for Methyl tert-butyl ether (MTBE). Available on the Internet at: http://cfpub.epa.gov/iristrac/. Last modified May 11, 2006.

USEPA. 2006b. *TRI Explorer: Trends*. Searches for MTBE. Available on the Internet at: http://www.epa.gov/triexplorer/trends.htm. Accessed February 8, 2006. Last modified June 8, 2005.

USEPA. 2006c. *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List*. EPA Report 815-D-006-007. Draft. December 2006.

USGS. 2003. Data sets for the random and focused surveys. Available on the Internet at: http://sd.water.usgs.gov/nawqa/vocns/nat_survey.html.

U.S. Water News Online. 2004. "Arizona governor signs bill to end use of gas additive MTBE." June. Available on the Internet at: http://www.uswaternews.com/archives/arcpolicy/4arizgove6.html.

Vaughan, V. 2005. "Valero to Quit Making Additive." MySanAntonio.com Business News. August 2. Available on the Internet at: http://www.mysanantonio.com/business/stories/MYSA080305.01E.Valero.12325438.html. Accessed October 7, 2005.

Washington State Department of Ecology (Washington DOE). 2000. *Occurrence of Methyl Tertiary-Butyl Ether (MTBE) in Groundwater at Leaking Underground Storage Tank Sites in Washington*. October 2000, Publication Number 00-09-054. Available on the Internet at: http://www.ecy.wa.gov/programs/tcp/ust-lust/MTBE_Report.pdf.

Weiser, M. 2004. Glennville forms water company. The Bakersfield Californian, September 28, 2004. Available on the Internet at: http://www.csub.edu/~dbaron/Sci360B/Glennville.html. Accessed December 9, 2004.

Wicherski, B. 1999. *An evaluation of Methyl tert-Butyl Ether (MTBE) in groundwater at leaking underground storage tank sites in Idaho*. Idaho Division of Environmental Quality.

Available on the Internet at:
http://www.deq.state.id.us/water/data_reports/ground_water/mtbe_lust_sites_enitre.pdf.

Williams, P.R.D. 2001. MTBE in California drinking water: an analysis of patterns and trends. *Environmental Forensics*. 2:75-85.

Williams, P.R.D., L. Benton, and P.J. Sheehan. 2003. MTBE in California's drinking water: a comparison of groundwater versus surface water sources [abstract]. *Environmental Forensics.* 4(3):175-189. Available on the Internet at:
http://taylorandfrancis.metapress.com/openurl.asp?genre=article&issn=1527-5922&volume=4&issue=3&spage=175.

Williams, P.R.D., L. Benton, and P.J. Sheehan. 2004. The risk of MTBE relative to other VOCs in public drinking water in California. *Risk Analysis.* 24(3):621-634.

Wilson, R.D., D.M. Mackay, and K.M. Scow. 2002. In Situ MTBE Biodegradation Supported by Diffusive Oxygen Release. *Environmental. Science & Technology*. 36:190-199.

Wood, S. 2002. Utility Will Get Millions from Oil Companies. Tahoe Daily Tribune and The Lake Tahoe News. August 6, 2002. Available at: http://www.tahoedailytribune.com.

Young, T.M. and R.D. Golding. 2002. Underground Storage Tank System Field-Based Research Project Report. Submitted to California State Water Resources Control Board. May 31. Available on the Internet at:
http://www.swrcb.ca.gov/cwphome/ust/leak_prevention/fbr/docs/FBR_Final_Report.pdf.

Zanardini, E., C. Pisoni, G. Ranalli, M. Zucchi, and C. Sorlini. 2002. Methyl tert-Butyl Ether (MTBE) Bioremediation Studies. *Annals of Microbiology.* 52:207-221.

Zogorski, J.S., A.M. Murduchowitz, A.L. Baehr, B.J. Bauman, D.L. Conrad, R.T. Drew, N.E. Korte, W.W. Lapham, J.F. Pankow, and E.R. Washington. 1997. "Fuel oxygenates and water quality," Chapter 2 of *Interagency Assessment of Oxygenated Fuels*. Washington, D.C.: Office of Science and Technology Policy, The Executive Office of the President. Available on the Internet at: http://www.epa.gov/otaq/regs/fuels/ostp-2.pdf.

Chapter 15:
Microorganisms on the CCL 2

A chapter from:

Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)

EPA Report 815-D-06-007

Contents

Exhibits

Abbreviations

AWWARF	American Water Works Association Research Foundation
CCL	Contaminant Candidate List
CCL 2	Second Contaminant Candidate List
CFU	Colony Forming Unit
DRINK	Drinking Water Research Information Network
DNA	Deoxyribonucleic Acid
ELISA	Enzyme Linked Immunosorbent Assay
FISH	Fluorescent In Situ Hybridization
GAC	Granular Activated Carbon
HPLC	High Performance Liquid Chromatography
IARC	International Association for Research on Cancer
ICR	Information Collection Rule
LC/MS	Liquid Chromatography/ Mass Spectrometry
LPS	Lipopolysaccharide
MAC	Mycobacterium Avium Complex
PAC	Powdered Activated Carbon
PCR	Polymerase Chain Reaction
PPIA	Protein Phosphatase Inhibition Assay
RNA	Ribonucleic Acid
UCMR	Unregulated Contaminant Monitoring Regulation
UCMR 1	First Unregulated Contaminant Monitoring Regulation
UV	Ultraviolet
WHO	World Health Organization

15 Microorganisms on the CCL 2

The nine microbial contaminants listed on the Second Contaminant Candidate List (CCL 2) include:

- Four virus groups - Caliciviruses, Echoviruses, Coxsackieviruses, and Adenoviruses
- Four bacteria/bacterial groups - *Aeromonas hydrophila; Helicobacter pylori; Mycobacterium avium intercellulare* or MAC; and Cyanobacteria (blue-green algae), other fresh water algae, and associated toxins
- One group of protozoa - Microsporidia (*Enterocytozoon bieneusi* and *Septata intestinalis*, now renamed *Encephalitozoon intestinalis*)

15.1 Evaluation of Microbial Contaminants for Regulatory Determination

In addition to considering if the Agency had sufficient information to address the three statutory criteria (i.e., adverse health effects, known/likely occurrence, and meaningful opportunity for health risk reduction), the Agency also considered whether sufficient information was available to determine the effectiveness of current treatment requirements for controlling the nine microbial contaminants. After consideration of these factors, the Agency determined that none of the nine microbial contaminants have sufficient information at this time to address the three statutory criteria or the question about whether current treatment practices adequately control for these organisms.

General areas where information is insufficient are identified in Exhibit 15-1. Section 15.2 briefly summarizes the available occurrence, health, analytical methods, and treatment information on the nine CCL 2 microorganisms. Section 15.3 provides a brief overview of ongoing research and data gaps.

Exhibit 15-1: Information Gaps for the CCL 2 Microbial Contaminants

Health Effects	Treatment	Analytical Methods	Occurrence
Microsporidia Some Cyanotoxins	*Aeromonas* MAC *Helicobacter* Adenoviruses Caliciviruses Coxsackieviruses Echoviruses Microsporidia Some Cyanotoxins	*Aeromonas* MAC *Helicobacter* Microsporidia Some Cyanotoxins	*Aeromonas* MAC *Helicobacter* Adenoviruses Caliciviruses Coxsackieviruses Echoviruses Microsporidia Some Cyanotoxins

15.2 Microbial Contaminant Profiles

15.2.1 Helicobacter pylori

Characteristics

Helicobacter pylori, first isolated from humans in 1983, is a spiral-shaped, microaerophilic, non-sporulating, gram-negative rod-shaped bacteria (CDC, 1999). Humans are the only know reservoir for *H. pylori*, which resides in the gastric mucous layer, or adheres to the epithelial lining of the stomach (Madigan *et al.*, 1997; CDC, 2002). In the United States, *H. pylori* is most prevalent among older adults, African Americans, Hispanics, and lower socioeconomic groups; about two-thirds of the world's population is infected (Staat *et al.*, 1996; CDC, 2002).

Health Effects

Peptic ulcer disease, gastric cancer, and gastric lymphoma are the classic health effects ascribed to *H. pylori* infection, but clinical manifestation may also include non-ulcer dyspepsia, and gastro-esophageal reflux disease (Vassili and Malfertheiner, 2003; Isakov and Malfertheiner, 2003). Recent evidence also suggests a possible association of cardio- and cerebrovascular diseases, hematologic disease, skin diseases, intractable nausea during pregnancy, and hepatobiliary disease with *H. pylori* infection (Gasbarrini *et al.*, 2003; Zuberbier, 2003; Diaz *et al.*, 2003). The International Association for Research on Cancer (IARC) has classified *H. pylori* as carcinogenic to humans (group 1) (IARC, 1994).

Analytical Methods

H. pylori is difficult to culture. A selective medium that discriminates it from background bacteria and a reliable method to detect viable organisms are under development (Degnan *et al.*, 2003). Molecular methods, such as fluorescent in situ hybridization (FISH) and the polymerase chain reaction (PCR), can aid in the detection of *H. pylori* but they fail to distinguish viable and non-viable organisms (Van Doorn *et al.*, 2000; Moreno *et al.*, 2003).

Occurrence and Exposure

The organism's only natural ecological niche is the stomach lining of humans. Evidence of this pathogen's survival in the environment is limited due to difficulty in culturing. Using molecular techniques, researchers have detected *H. pylori* in ambient water, including some drinking water sources, in the U.S., Canada, Japan, and Sweden (Hegarty *et al.*, 1999a; McKeown *et al.*, 1999; Sasaki *et al.*, 1999; Hulten *et al.*, 1998). More research is needed on the occurrence of viable *H. pylori* in drinking water, including ground water sources that may be affected by ambient surface waters. *H. pylori* is included on List 3 of EPA's Unregulated Contaminant Monitoring Rule (UCMR) (64 FR 50556). Monitoring will begin when a suitable analytical method has been developed and tested.

H. pylori has a low infective dose and a high prevalence in human populations. Seroprevalence studies indicate that more than 50 percent of people in the United States are

infected, although this rate is declining (Staat *et al.*, 1996). Some epidemiological data on *H. pylori* suggest host-to-host transmission, although common sources of infection, such as food or drinking water, have also been implicated (Malaty *et al.*, 1991; Blecker *et al.*, 1994). The likelihood of waterborne transmission in the U.S. has not been determined, but it is a strong risk factor in developing countries (Hegarty *et al.*, 1999b). Most infections are acquired in childhood, although children may remain asymptomatic (Lanciers *et al.*, 1996; Rowland and Drumm, 1998).

Water Treatment

Studies indicate that individual cases of *H. pylori* are readily inactivated by chlorine and removed by conventional and membrane filtration (Johnson *et al.*, 1997; Gerba *et al.*, 2003a). However, there is uncertainty about the survival rates of the non-culturable coccoid form. Also, there is still a question about the disinfection efficiency of disinfectants other than chlorine, especially for aggregated or adsorbed cells (Baker *et al.*, 2002).

15.2.2 Aeromonas hydrophila

Characteristics

Aeromonas hydrophila is a gram-negative, oxidase-positive, non-spore-forming, facultatively anaerobic rod, some strains of which are of clinical importance. *A. hydrophila* is free-living in soil and water, and is not necessarily associated with fecal contamination. It grows in the biofilm of distribution systems.

Health Effects

Aeromonas species can cause gastroenteritis and infection of wounds (Smith and Cheasty, 1998; Janda and Abbott, 1998; Vila *et al.*, 2003). Diarrhea is generally self-limited and lasts a few days to a few weeks (Tomar, 2001). Chronic diarrhea of more than a few weeks has been described in children under the age of five years who have predisposing conditions such as treatment with antibiotics to which Aeromonads are resistant (Moyer, 1987). Disseminated, systemic disease in the compromised hosts (especially those who have underlying liver disease or cancer) causes a high fatality rate. Other systemic infections include bacteremia, septicemia, cirrhosis, and endocarditis (Lau *et al.*, 2000; Chang *et al.*, 1997; Braun *et al.*, 2001; Brouqui and Rault, 2001).

Exposure to *Aeromonas* occurs through ingestion of contaminated food and water, and dermal contact with water or soil. Person-to-person transmission is rare (Farmer *et al.*, 1992; Janda and Abbott, 1998). Transmission generally occurs by exposure to *Aeromonas* in aqueous environments, either via trauma or wound infection or by ingestion (Altwegg and Geiss, 1989; Esteban *et al.*, 1999). Exposure to surface water is the primary risk factor associated with wound infection. No waterborne outbreaks have been reported.

Analytical Methods

EPA Method 1605 uses a selective medium to distinguish species and strains of Aeromonads in water (USEPA, 2000). This was the method used for screening under the UCMR. One drawback of this method is that it only identifies to the genus level and thus does not differentiate between pathogenic and non-pathogenic strains (Altwegg *et al.*, 1990). Several molecular methods are available but none have been standardized for general use (Peng *et al.*, 2002; Wang *et al.*, 2003).

Occurrence and Exposure

A. hydrophila has been found in all types of water: wastewater, surface water, ground water, marine and estuarine environments, and even chlorinated water supplies (Pettibone, 1998; Borrell *et al.*, 1998; Bianucci *et al.*, 2001). It grows in distribution systems (Gavriel *et al.*, 1998; Smith and Cheasty, 1998). EPA included *Aeromonas* as an analyte in the recent UCMR Screening Survey (2001-2003). Under this survey, drinking water samples were collected from 300 water systems, 6 times a year, and at 3 different sampling locations per system. The samples were analyzed using EPA Method 1605 (USEPA, 2000). *Aeromonas* was detected in 2.6% of the samples. (For details, see the forthcoming report entitled *The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List.*) Further work is needed to determine the pathogenicity of isolates detected in these samples.

Water Treatment

Standard water treatment with chlorine disinfectant appears to reduce the numbers of Aeromonads to levels below 1 colony forming unit (CFU) per 100 ml (Nichols, 1996). However, treated water can support Aeromonad growth in storage reservoirs or distribution systems, and Aeromonads can contribute to biofilm, which makes them more resistant to disinfectant residuals (Gavriel *et al.*, 1998). The concentration and rate of growth depend on temperature, organic content of the water, residence time in the distribution system, and the amount of residual chlorine disinfectant (Burke *et al.*, 1984).

15.2.3 Mycobacterium avium Complex (MAC)

Characteristics

MAC is a group of slow-growing, gram-positive, aerobic, rod-shaped bacteria that consists of two predominant species, *Mycobacterium intracellulare* and *Mycobacterium avium*, the latter of which has three subspecies: *M. avium* ssp. *avium*, *M. avium* ssp. *paratuberculosis*, and *M. avium* ssp. *silvaticum* (Thorel *et al.* 1990a, 1990b; Inderlied *et al.* 1993; Cangelosi *et al.* 2003). These bacteria are ubiquitous in the environment and cause opportunistic infections in humans and animals (Falkinham, 1996). Due to the low permeability of their cell walls, MAC are resistant against therapeutic agents (Minnikin, 1991; Nikaido *et al.*, 1993). Mycolic acids, complex lipids located within the cell wall, contribute to the hydrophobic nature of the cell envelope and are thought to play a role in the organism's resistance against therapeutic agents (Nikaido *et al.*, 1993).

Health Effects

MAC infections occur most often in immunocompromised individuals (Horsburgh, 1991). Infections caused by MAC include cervical lymphadenitis (inflammation of neck lymph nodes), joint infections, pulmonary infections and bacteremia (Swanson *et al.*, 1998; Wolinsky, 1995; Horsburg *et al.*, 1992). MAC-related pulmonary disease typically occurs in patients with impaired cellular immunity or chronic lung disease (Aksamit, 2002). MAC usually manifests in AIDS patients as a disseminated disease involving the lungs, lymph nodes, and gastrointestinal tract. Mycobacterial cervical lymphadenitis has long been recognized as a disease of children between 6 months and two years of age; infection is limited to the cervical and mandibular lymph nodes. *M. paratuberculosis* is suspected in the etiology of Crohn's Disease (Romero *et al.*, 2005). EPA established a Health Advisory for mycobacteria in 1999 (USEPA, 1999).

Analytical Methods

Culture and isolation of MAC bacteria from environmental samples is problematic because of their slow growth (it could take months to see colonies on plates), particular nutrient requirements, and the presence of other microorganisms that quickly outgrow MAC. The use of nucleic acid probes for group or species determination has generally replaced biochemical identification methods for screening samples. Definitive identification of mycobacteria is possible by determination of methylated fatty acid or through examination of mycolic acid profiles by chromatographic methods (Ozbek and Aktas, 2003; HPLC Users Group, 1999). PCR methods are available to facilitate direct detection of MAC in environmental samples. Molecular typing methods have demonstrated remarkable heterogeneity among MAC strains; typing has shown both taxonomic and epidemiological value (van Soolingen, 2001).

Occurrence and Exposure

MAC has been isolated from tap water samples worldwide (Aronson *et al.*, 1999; Covert *et al.*, 1999). The presence of these bacteria in tap water is attributed to their high resistence to disinfectants commonly used in water treatment and their ability to grow in biofilms in distribution systems (von Reyn *et al.*, 1993, 1994). MAC is thermo-tolerant (it grows at temperatures in the range of 52 to 57 ^{o}C), and has frequently been isolated from recirculated hot water systems in institutions such as hospitals (Embil *et al.*, 1997; Kahana and Kay, 1997). Increased zinc levels have been suggested to favor growth and survival of MAC. Some hospitals use galvanized pipes made with zinc alloys which would contribute to MAC growth. In several studies, MAC has been isolated from biofilm in water distribution systems in the U.S., indicating that biofilms may be a significant reservoir for the organism (Norton *et al.*, 1999; Falkinham *et al.*, 2001).

Water Treatment

MAC bacteria exhibit significant resistence to chlorine, chloramine, chlorine dioxide, and ozone disinfection of drinking water (Taylor *et al.*, 2000; Le Dantec *et al.* 2002; Gerba *et al.*, 2003a). This is probably due to the presence of waxy material in their cell wall and the ability of some strains to clump together (Gerba *et al.*, 2003a). However, because mycobacteria adhere to

particles, a large fraction are removed from drinking water by flocculation, sedimentation, and filtration. They are also sensitive to ultraviolet radiation (USEPA, 2002).

MAC bacteria have been isolated from drinking water distribution biofilms (Iivanainen *et al.*, 1999; Falkinham *et al.*, 2001). The fact that colonization of water distribution systems occurs in both surface and ground water sources suggests that mycobacteria in the distribution system might represent renewable biofilm (Falkinham, 2002).

15.2.4 Cyanobacteria (blue-green algae), other fresh water algae, and their toxins

Characteristics

Cyanobacteria, often referred to as blue-green algae, more closely resemble bacteria than algae. Of the thousands of cyanobacterial species that are known to exist, an estimated 46 of them produce toxins (WHO, 1998). Cyanobacterial toxins are divided into three groups: cyclic peptides, alkaloids, and lipopolysaccharides (LPSs). The toxins of most concern in the United States are microcystin, which is a cyclic peptide, and cylindrospermopsin, anatoxin-a, saxitoxin, and anatoxin-a(s), which are all alkaloids (USEPA, 2001).

Health Effects

Most recognized cyanotoxins are hepatotoxins or neurotoxins. Cyanobacterial toxins can also cause skin irritation, acute gastroenteritis, and possibly cancer (Chorus and Bartram, 1999). Illnesses have been reported following ingestion of contaminated water, and in at least one case deaths occurred after hemodialysis treatments that used water contaminated with cyanotoxins (130 patients became ill, and 50 of them died). Biological and chemical evidence pointed to the occurrence of microcystins in treatment water as being the major factor in these deaths (Carmichael *et al.*, 2001; Pouria *et al.*, 1998; Jochimsen *et al.*, 1998; Chorus and Bartram, 1999). Children and immunocompromised people may be at greatest risk of health effects from cyanobacterial toxins (Pilotto *et al.*, 1999). The World Health Organization (WHO, 2004) has set a provisional guideline value of 1 µg/L for microcystin-LR.

Analytical Methods

Several analytical methods are available for detecting cyanobacteria and their toxins in drinking water. Some research methods currently available for some toxins include high performance liquid chromatography (HPLC), enzyme linked immunosorbent assay (ELISA), protein phosphatase inhibition assay (PPIA), and liquid chromatography/mass spectrometry (LC/MS). However, none of these methods have been standardized (USEPA, 2001).

Occurrence and Exposure

Cyanobacteria are ubiquitous in the aquatic environment. They occur in surface water whenever nutritional, temperature, and water flow conditions are favorable for their growth (Horne and Goldman, 1994; Yoo *et al.*, 1995; Chorus and Bartram, 1999). Some cyanobacteria release toxins throughout their life cycle, while others only release toxins upon cell death or lysis (Yoo *et al.*, 1995). When released to the aquatic environment, cyanobacterial toxins may persist

for several weeks to months (Yoo *et al.*, 1995; Chorus and Bartram, 1999). The American Water Works Association Research Foundation (AWWARF, 2000) performed a preliminary monitoring study of microcystins in drinking water using a screening method. The result showed that 80% of the samples (539 out of 677) collected by participating U.S. and Canadian water utilities were positive for microcystins but only 4.3% of the positive samples had concentrations higher than WHO's 1 µg/L guideline. A recent Wisconsin study showed microcystin levels to be as high as 6 µg/L in some raw waters (USEPA, 2001). At this time there is no national database on the occurrence and frequency of cyanobacterial blooms or their toxins in the United States.

Water Treatment

Controlling source water to make it unsuitable for algal growth is one of the most effective methods of water treatment (Burns, 2000; USEPA, 2001). Riverbank filtration also shows promise for toxin removal (Holst *et al.*, 2003). Water treatment techniques (coagulation, sedimentation, filtration, disinfection, granular activated carbon (GAC), powdered activated carbon (PAC), ozonation, and ultraviolet radiation) are effective to varying degrees at removing most of the most common cyanobacteria and their toxins in drinking water (USEPA, 2001; Burns, 2000; Hitzfeld *et al.*, 2000). When the appropriate combination of techniques is used, close to 100 percent of particular toxins can generally be eliminated in finished water, though algal blooms and high organic loads can limit treatment effectiveness (Drikas *et al.*, 2001; Karner, *et al.*, 2001; Hitzfeld *et al.*, 2000). Some treatment options (e.g., copper sulfate application) can cause cell lysis and produce an increase in extracellular toxin concentrations (Yoo *et al.*, 1995; USEPA, 2001).

15.2.5 Adenoviruses

Characteristics

Adenoviruses are double-stranded deoxyribonucleic acid (DNA) viruses. These are the largest of the viruses on the Contaminant Candidate List (CCL). They range in size between 60 and 90 nm (Foy, 1997). Adenoviruses were first noted in human adenoid and tonsil tissue in 1953 (Rowe *et al.*, 1953). More than 50 adenovirus types exist (De Jong *et al.*, 1999). Different types cause a wide range of health effects. Because adenoviruses can occur in human feces (Pina *et al.*, 1998), there is a potential for waterborne transmission. Adenovirus types with the greatest such potential are the enteric adenoviruses, types 40 and 41 (Foy, 1997).

Health Effects

Children under two years old are especially vulnerable to enteric adenovirus infection (LeBaron *et al.*, 1990). The immunocompromised are another sensitive subpopulation for adenoviruses (Hierholzer, 1992). Adenovirus infection can be present in the immunocompromised as a disseminated disease, affecting many different parts of the body. Adenoviruses can cause respiratory tract infection, pharyngitis, conjunctivitis, cystitis, gastroenteritis and other effects (Foy, 1997). Other serious health effects include Reyes Syndrome (Edwards *et al.*, 1985) and myocarditis (Pauschinger *et al.*, 1999). Adenoviruses have also been associated with weight gain in animals (Dhurandhar *et al.*, 2000).

Analytical Methods

No standardized and validated method exists for detecting adenoviruses in water. Adenoviruses grow in tissue culture cells, although the enteric adenoviruses, types 40 and 41, require different cell lines from the other adenovirus types. Adenoviruses grow slowly and may be overgrown by other viruses in mixed samples (Hurst *et al.*, 1988). A variety of molecular techniques involving PCR amplification have been used for detection and identification of adenoviruses (Chaperon *et al.*, 2000; Jiang *et al.*, 2001; Loge *et al.*, 2002).

Occurrence and Exposure

Adenoviruses are among the most commonly detected virus types in environmental waters (Chaperon *et al.*, 2000). Adenovirus infection by exposure to recreational water is well documented (D'Angelo *et al.*, 1979; Turner *et al.*, 1987; Papapetropoulou and Vantarakis, 1998). However, no cases of infection via drinking water have been reported. In some cases, researchers have observed seasonal variation in the frequency of diseases caused by adenoviruses (Krikelis *et al.*, 1985; Tani *et al.*, 1995). Adenoviruses appear to survive better in some water types than other viruses. Researchers have speculated that the greater longevity of adenoviruses in water may be due to their reliance on DNA, which is more stable than ribonucleic acid (RNA) (Enriquez *et al.*, 1995).

Water Treatment

Adenoviruses can be controlled by chlorine disinfection in laboratory studies (Gerba, 2003a). Aggregation of virus and adherence to particles can reduce the effectiveness of chlorine disinfection (Payment *et al.*, 1985). Adenoviruses are much more resistant to ultraviolet (UV) irradiation than other viruses (Gerba *et al.*, 2003a).

15.2.6 Caliciviruses

Characteristics

Caliciviruses are viruses that belong to the family *Caliciviridae*. The first documented outbreak of Norwalk virus, the best-known of the caliciviruses, occurred in Norwalk, Ohio in 1968 (Adler and Zicki, 1969). The Norwalk virus and related strains, known collectively as noroviruses, present a potential health risk in drinking water in the United States. The noroviruses are single-stranded RNA viruses (Jiang *et al.*, 1990). They are about 25 to 35 nm in diameter. Noroviruses occur in feces and have caused many waterborne disease outbreaks.

Health Effects

Human caliciviruses, including noroviruses, cause a self-limiting gasteroenteritis which usually lasts 24 to 48 hours. Symptoms include vomiting, abdominal cramps, and diarrhea (MMWR, 1990).

Analytical Methods

No cell culture method for noroviruses exists (Atmar and Estes, 2001). Thus, viable noroviruses can't be enumerated in water or in treatment studies. Molecular detection methods are available, but no molecular method has yet been standardized for detection of noroviruses in water.

Occurrence

Occurrence information for noroviruses in water is limited due to the inadequacies of analytical methods. Nonetheless, noroviruses have been linked to many waterborne disease outbreaks in the U.S. (Kaplan *et al.*, 1982; Parshionikar *et al.* 2003). They have been detected in sewage effluent (Lodder *et al.*, 1999) and in ambient water (Griffin *et al.*, 1999).

Water Treatment

Studies of the susceptibility of human caliciviruses to water treatment are limited by the lack of culture methods for detecting viable viruses. Nevertheless, Shin *et al.* (1998) determined that Norwalk virus was much less resistant to chlorine inactivation than poliovirus, and was inactivated about as rapidly as MS2 bacteriophage. The small size of noroviruses may make them difficult to remove by filtration, unless they are aggregated with larger particles.

15.2.7 Echoviruses and Coxsackieviruses

Characteristics

Echoviruses and coxsackieviruses are single-stranded RNA viruses that are between 27 and 30 nm in diameter. Both viruses belong to the genus *Enterovirus*. Echoviruses were first isolated from cell cultures in the late 1940s. Coxsackieviruses were first isolated from patients with polio-like symptoms in Coxsackie, New York in 1948. There are at least 31 human echoviruses and 29 human coxsackieviruses. These viruses are transmitted through the fecal-oral route.

Health Effects

Coxsackieviruses and echoviruses first infect the intestinal tract, with or without symptoms (Modlin, 1986; Minor, 1998). After entering the blood stream, different serotypes of the viruses can infect most other organ systems. They may cause febrile illness, aseptic meningitis, respiratory disease, encephalitis, paralytic disease, and other effects (Melnick, 1997). Some serotypes can cause myocarditis (Martino *et al.*, 1995). Others are suspected of causing diabetes (Bantvala *et al.* 1985; Craighead, 1975; Notkins, 1977). Echovirus and coxsackievirus infections are among the most common viral infections of humans in the United States (CDC, 1997).

Analytical Methods

The Information Collection Rule (ICR) method is a standardized and validated method for the detection of culturable echoviruses and coxsackieviruses. However, the ICR method does not differentiate virus types. A PCR-based method can be used to differentiate coxsackieviruses and echoviruses detected in water (Vivier *et al.* 2001). However, this method does not necessarily indicate which viruses are infective. A hybrid method, the integrated cell culture/PCR-based method described by Reynolds *et al.* (2001), demonstrates viability through cell culture, but also gives PCR detection.

Occurrence

Coxsackieviruses and echoviruses are shed in feces and occur in sewage and contaminated waters. These viruses have been detected in ambient water (Vivier *et al.*, 2001), ground water (Powell *et al.*, 2003), and drinking water (Vivier *et al.* 2004). Waterborne outbreaks of disease caused by enteroviruses are not common, but have been documented (Hejkal *et al.* 1982).

Water Treatment

Hurst (1991) reported that physical water treatment with disinfection was effective in removing enteroviruses. Studies by Sobsey *et al.* (1988), Englebrecht *et al.* (1980), Payment *et al.* (1985), and others suggest that both echoviruses and coxsackieviruses are susceptible to chlorine disinfection. Nonetheless, culturable enteroviruses have been detected in treated water (Vivier *et al.*, 2004). Also, questions remain about the effectiveness of disinfection against particle-bound viruses, since the particles can afford some protection for viruses (Hejkal *et al.*, 1981).

15.2.8 Human Microsporidia: Enterocytozoon bieneusi and Encephalitozoon (formerly Septata) intestinalis

Characteristics

Microsporidia were first recognized as a distinct group of microorganisms in 1882 (Wittner, 1999). Microsporidia were first reported as human pathogens in 1959 (Matsubayashi *et al.*, 1959), and since 1985 they have emerged as pathogens of concern in immunocompromised patients (Desporte *et al.*, 1985). The two species of greatest concern for the immunocompromized are *Enterocytozoon bieneusi* and *Encephalitozoon intestinalis*. Microsporidia have an environmentally resistant stage, the spore, which gets its survival characteristics from its impervious spore wall. The spore is the infectious stage for new hosts. The spores of the human microsporidia species of concern are very small, in the range of 1 to 3 µm, as compared to *Cryptosporidium* spp. oocysts, which are 4 to 6 µm (Franzen and Müeller, 1999).

Health Effects

Microsporidiosis primarily affects individuals who are immunocompromised, especially those infected with HIV (Bryan, 1995). Weiss and Keohane (1997) estimated that as many as 50% of individuals infected with HIV suffer from microsporidiosis. However, Kotler and Orenstein (1999) find that the recent success of HIV-control drugs in boosting immune function has greatly reduced the incidence of microsporidiosis in HIV-infected patients.

In the immunocompromised, microsporidia infect the gastrointestinal tract, causing diarrhea (Hutin *et al.*, 1998). They may also become disseminated throughout the body in these patients, causing a variety of adverse health effects (Orenstein *et al.*, 1997). Microsporidiosis appears to be uncommon in the immunocompetent. When it does occur, the most common effect in the immunocompetent appears to be a self-limiting diarrhea (Bryan *et al.*, 1997). Cases of traveler's diarrhea have been attributed to microsporidiosis (Fournier *et al.*, 1998; Sobottka *et al.*, 1995).

Analytical Methods

There is no standardized and validated method for detecting human microsporidia in environmental waters or drinking water. Research methods have been used to detect microsporidia in surface waters (Sparfel *et al.*, 1997; Fournier *et al.*, 2000) and ground waters (Dowd *et al.*, 1998). However, these methods have limitations. Methods involving water concentration, as described for example by Sparfel *et al.* (1997) and Borchardt and Spencer (2002), are not widely available. Due to the small size and lack of internal structure of microsporidia, direct microscopic detection is not a viable option. Methods that have been used in various microsporidia studies include PCR (Sorel *et al.*, 2003), real-time PCR (Menotti *et al.* 2003), and fluorescent in-situ hybridization (Hester *et al.*, 2000). Development of analytical methods for *E. bieneusi* is hindered by the lack of a culture method capable of producing spores in sufficient quantities for testing. A culture method is available for *Encephalitozoon* species (Bouladoux *et al.*, 2003).

Occurrence and Exposure

Due to the fecal and urinary mode of shedding of microsporidian spores (Orenstein *et al.*, 1992), waterborne transmission is possible. The spores of microsporidia can survive for weeks or months, and some can also survive drying (Kramer, 1970; Maddox, 1973; Undeen *et al.*, 1993). *E. bieneusi* and *E. intestinalis* species have been detected in sewage (Franzen and Müeller, 1999), surface water (Sparfel *et al.*, 1997), ground water (Dowd *et al.*, 1998), and irrigation water (Thurston *et al.*, 1999). They have not been detected in drinking water. *E. bieneusi* and *E. intestinalis* have been found in a variety of domestic and wild animals (Rinder *et al.*, 1997). In human hosts, there is no evidence to indicate a seasonality of microsporidiosis (Conteas *et al.*, 1998).

Water Treatment

Data on the effectiveness of water treatment for controlling *E. bieneusi* are limited due to the lack of a culture method for this organism. Since *Encephalitozoon* species are culturable,

their susceptibility to treatment has been studied. In a model treatment plant, conventional physical treatment provided 2.47 log removal of *E. intestinalis* (Gerba *et al.*, 2003b). *E. intestinalis* is somewhat resistant to chlorine, but can nonetheless be controlled (Wolk *et al.*, 2000). *E. intestinalis* is also susceptible to UV radiation and ozone (Naumovitz *et al.*, 1998).

15.3 On-Going Research Activities at EPA to Overcome Data Gaps for the CCL 2 Microorganisms

EPA supports an active research program on the CCL 2 microorganisms to fill information gaps. For the design of treatment studies on the CCL viruses or surrogates, EPA emphasizes the need to conduct tests under realistic conditions, e.g., conditions where viruses might be protected by aggregating or adhering to particles. EPA believes it is important to conduct virus removal/inactivation studies in drinking water treatment plants or pilot plants. EPA is also pursuing method development for viruses to support these treatment studies.

EPA has completed a one-year UCMR survey of the genus *Aeromonas* in 292 public water systems. Researchers are still working on ways to characterize clinical strains and distinguish them from non-pathogenic strains, and developing methods to detect *Aeromonas* virulence factors. Similarly, researchers have conducted drinking water surveys for MAC, but they are still working on refining analytical methods and characterizing virulence factors. Methods for *H. pylori* are under development.

EPA is currently investigating the susceptibility of microsporidian *E. intestinalis* to chlorine and chloramine. In addition, EPA is sponsoring methods-related work on fluorescent gene probes, real-time PCR, concentration methods, and immunomagnetic separation for microsporidia. As part of ongoing environmental monitoring, researchers recently confirmed the presence of microsporidia in ground water. Researchers also participated in a workshop to assess the status of work on microsporidia.

EPA has funded projects on the removal of algal cells and cyanotoxins in a pilot-scale treatment plant, and on the effect of disinfection on cyanotoxins. EPA has developed analytical chemistry cyanotoxin standards, and is currently developing analytical methods for potential use in future monitoring. EPA has conducted several occurrence surveys for cyanotoxins and also a number of health effects studies. Researchers are currently conducting risk assessments to determine reference doses for the cyanotoxins. EPA has organized and participated in several workshops on cyanotoxins to assess the state of the science. EPA is taking steps to establish infective doses for CCL microbial contaminants through a formal review process.

For further information on these projects, please refer to the EPA's Office of Ground Water and Drinking Water Research Information Network (DRINK), found at www.epa.gov/safewater/drink/intro.html, a publicly accessible, web-based system that tracks over 1,000 ongoing research projects.

15.4 References

Adler, J.L. and R. Zicki. 1969. Winter vomiting disease. *Journal of Infectious Diseases*. 119:668-673.

Aksamit, T. R. 2002. Mycobacterium avium complex pulmonary disease in patients with pre-existing lung disease. *Clinics in Chest Medicine*. 23(3):643-653.

Altwegg, M. and H.K. Geiss. 1989. Aeromonas as a human pathogen. *CRC Critical Reviews in Microbiology*. 16: 253-286.

Altwegg, M., A.G. Steigerwalt, R. Altwegg-Bissig, J. Luthy-Hottenstein, and D.J. Brenner. 1990. Identification of Aeromonas genospecies from humans. *Journal of Clinical Microbiology*. 28(2): 258-264.

American Water Works Association Research Foundation (AWWARF). 2000. Assessment of Blue-Green Algal Toxins in Raw and Finished Drinking Water. Prepared by Wayne W. Carmichael.

Aronson, T., A. Holtzman, N. Glover, M. Boian, S. Froman, O. G. Berlin, H. Hill, and G. Stelma, Jr. 1999. Comparison of large restriction fragments of Mycobacterium avium isolates recovered from AIDS and non-AIDS patients with those of isolates from potable water. *Journal of Clinical Microbiology*. 37(4):1008-1012.

Atmar, R.L. and M.K. Estes. 2001. Diagnosis of noncultivatable gasteroenteritis viruses, the human caliciviruses. *Clinical Microbiology Reviews*. 14(1):15-37.

Baker, K.H., J.P. Hegarty, B. Redmond, N.A. Reed, and D.S. Herson. 2002. Effect of Oxidizing Disinfectants (Chlorine, Monochloramine, and Ozone) on Helicobacter pylori. *Applied and Environmental Microbiology*. 68(2):981-984.

Bantvala, J.E., G. Schernthaner, E. Schober, M.L. De Silva, J. Bryant, M. Borkenstein, D. Brown, M.A. Menser, and M. Silink. 1985. Cocksackie B, mumps, rubella, and cytomegalovirus specific IgM responses in patients with juvenile-onset insulin-dependent diabetes mellitus in Britain, Austria, and Australia. *Lancet.* 1:1409-1412.

Bianucci, F., M. Bernagozzi, R. Sacchetti, and P. Bisbini. 2001. [Determination of Aeromonas spp. and Pseudomonas spp. in chlorinated water supply]. *Annali di Igiene.* 13(3):185-189.

Blecker, U., S. Lanciers, E. Keppens, and Y. Vandenplas. 1994. Evolution of Helicobacter pylori positivity in infants born from positive mothers. *Journal of Pediatric Gastroenterology and Nutrition.* 19: 87-90.

Borchardt, M.A. and S.K. Spencer. 2002. Concentration of Cryptosporidium, microsporidia and other water-borne pathogens by continuous separation channel centrifugation. *Journal of Applied Microbiology*. 92(4): 649-656.

Borrell, N., M.J. Figueras, and J. Guarro. 1998. Phenotypic identification of Aeromonas genomospecies from clinical and environmental sources. *Canadian Journal of Microbiology.* 44:103-108.

Bouladoux, N., S. Biligui, and I. Desportes-Livage. 2003. A new monoclonal antibody enzyme-linked immunosorbent assay to measure in vitro multiplication of the microsporidian Encephalotizoon intestinalis. *Journal of Microbiological Methods.* 53(3):377-385.

Braun, P., G. Balzer, and K. Fehlhaber. 2001. Activity of bacterial lipases at chilling temperatures. *Food Microbiology* (London). 18(2):211-215.

Brouqui, P. and D. Raoult. 2001. Endocarditis due to rare and fastidious bacteria. *Clinical Microbiology Reviews.* 14(1):177-207.

Bryan, R.T. 1995. Microsporidiosis as an AIDS-related opportunistic infection. *Clinical Infectious Diseases* (suppl):S21-S65.

Bryan, R.T., D.A. Schwartz, and R. Weber. 1997. Microsporidiosis in *patients who are not infected with human immunodeficiency virus. Clinical* Infectious Diseases. 24:534-535.

Burke, V., J. Robinson, M. Gracey, D. Peterson, and K. Partridge. 1984. Isolation of Aeromonas hydrophila from a metropolitan water supply: seasonal correlation with clinical isolates. *Applied and Environmental Microbiology*. 48:361-366.

Burns, J. 2000. A Review of Reports and Meetings Regarding Cyanotoxins in Australian Drinking Water supplies, Human Health Guidelines, and Water Treatment Practices. In: Fleming L.E., C. Rivero, and J. Burns. Final Report: Blue Green Algal Bloom Taskforce, October 2000.

Cangelosi, G. A., J. Clark-Curtiss, M. A. Behr, T. Bull, and T. Stinear (ed.). 2003. Biology of pathogenic mycobacteria in water. Geneva: WHO and USEPA.

Carmichael, W.W., S.M.F.O. Azevedo, J.S. An, R.J.R. Molica, E.M. Jochimsen, S. Lau, K.L. Rinehart, G.R. Shaw, and G.K. Eaglesham. 2001. Human Fatalities from Cyanobacteria: Chemical and Biological Evidence for Cyanotoxins. *Environmental Health Perspectives*. 109:663-668.

Centers for Disease Control (CDC). 1997. Nonpolio enterovirus surveillance- United States, 1993-1996. Mor. Mortal. Wkly. Rep. CDC. *Surveillance Summaries*. 46(32):748-750.

CDC. 1999. Spiral bacteria in the human stomach. Available on the Internet at: http://www.cdc.gov/search.do?action=search&queryText=spiral+bacteria+&x=15&y=9. Accessed on May 3, 2005.

CDC. 2002. Helicobacter pylori and peptic ulcer disease - Fact sheet for healthcare providers. Available on the Internet at: http://www.cdc.gov/search.do?action=search&queryText=helicobacter&x=14&y=6. Accessed on May 3, 2005.

Chang, C. Y., H. Thompson, N. Rodman, J. Bylander, and J. Thomas. 1997. Pathogenic analysis of Aeromonas hydrophila septicemia. *Annals of Clinical & Laboratory Science*. 27(4):254-259.

Chaperon, C.D., N.A. Ballester, J.H. Fontaine, C.N. Frades, and A.B. Margolin. 2000. Detection of astroviruses, enteroviruses, and adenovirus types 40 and 41 in surface waters collected and evaluated by the information collection rule and an integrated cell culture-nested PCR procedure. *Applied and Environmental Microbiology*. 66(6):2520- 2525.

Chorus, I. and J. Bartram. (eds.) 1999. Toxic Cyanobacteria in Water: A Guide to Their Public Health Consequences, Monitoring and Management. World Health Organization. London: E & FN Spon.

Conteas, C.N., O.G. Berlin, M.J. Lariviere, S.S. Pandhumas, C.E. Speck, R. Porschen, and T. Nakaya. 1998. Examination of the prevalence and seasonal variation of intestinal microsporidiosis in the stools of persons with chronic diarrhea and human immunodeficiency virus infection. *American Journal of Tropical Medicine and Hygiene*. 58:559-561.

Covert, T. C., M. R. Rodgers, A. L. Reyes, and G. N. Stelma, Jr. 1999. Occurrence of nontuberculous mycobacteria in environmental samples. *Applied and Environmental Microbiology.* 65(6):2492-2496.

Craighead, J.E. 1975. The role of viruses in the pathogenesis of pancreatic disease and diabetes mellitus. *Progress in Medical Virology*. 19:161-214.

D'Angelo, L.J., J.C. Hierholzer, R.A. Keenlyside, L.J. Anderson, and W.J. Martone. 1979. Pharyngoconjunctival fever caused by adenovirus type 4: Report of a swimming pool-related outbreak with recovery of virus from pool water. *Journal of Infectious Diseases*. 140:42-47.

De Jong, J.C., A.G. Wermenbol, M.W. Verweij-Uijterwaal, K.W. Slaterus, P. Wertheim-Van Dillen, G.J.J. Van Doornum, S.H. Khoo, and J.C. Hierholzer. 1999. Adenoviruses from human immunodeficiency virus-infected individuals, including two strains that represent new candidate serotypes Ad50 and Ad51 of species B1 and D, respectively. *Journal of Clinical Microbiology*. 37(12):3940-3945.

Degnan, A.J., W.C. Sonzogni, and J.H. Standridge. 2003. Development of a Plating Medium for Selection of Helicobacter pylori from Water Samples. *Applied and Environmental Microbiology*. 69(5):2914-2918.

Desportes, I., Y. LeCharpentier, A. Galian, F. Bernard, B. Cochand-Priolett, A. Laverne, P. Ravisse, and R. Modigliani. 1985. Occurrence of a new microsporidian: Enterocytozoon bieneusi n.g., n.sp., in the enterocytes of a human patient with AIDS. *Journal of Protozoology*. 32:250-254.

Dhurandhar, N.V., B.A. Israel, J.M. Kolesar, G.F. Mayhew, M.E. Cook, and R.L. Atkinson. 2000. Increased adiposity in animals due to a human virus. *International Journal of Obesity*. 24:989-996.

Diaz, C., C.J. O'Callaghan, A. Khan, and A. Ilchyshyn. 2003. Rosacea: a cutaneous marker of Helicobacter pylori infection? Results of a pilot study. *Acta Dermato-Venereologia*. 83(4):282-286.

Dowd, S.E., C.P Gerba, and I.L. Pepper. 1998. Confirmation of the human-pathogenic microsporidia Enterocytozoon bieneusi, Encephalitozoon intestinalis, and Vittaforma corneae in water. *Applied and Environmental Microbiology*. 64(9):3332-3335.

Drikas, M., C.W.K. Chow, J. House, and M.D. Burch. 2001. Using coagulation, flocculation, and settling to remove toxic cyanobacteria. *Journal of the American Water Works Association*. 93(2):100-111.

Edwards, K.M., S.R. Bennett, W.L. Garner, D.L. Bratton, A.D. Glick, H.L. Greene, and P.F. Wright. 1985. Reye's syndrome associated with adenovirus infections in infants. *American Journal of Diseases of Children*. 139:343-346.

Embil, J., P. Warren, M. Yakrus, R. Stark, S. Corne, D. Forrest, and E. Hershfield. 1997. Pulmonary illness associated with exposure to Mycobacterium avium complex in hot tub water. Hypersensitivity pneumonitis or infection? *Chest*. 111(3):813-816.

Engelbrecht, R. S., M. J. Weber, B. L. Salter, and C. A. Schmidt. 1980. Comparative inactivation of viruses by chlorine. *Applied and Environmental Microbiology*. 40:249-256.

Enriquez, C.E., J. Sandoval-Garzon, and C.P. Gerba. 1995. Survival, detection, and resistance to disinfection of enteric adenoviruses. Proceedings of AWWA Water Quality Technology Conference. pp. 2059- 2086.

Esteban, J., I. Gadea, R. Fernandez-Roblas, A. Molleja, R. Calvo, V. Acebron, and F. Soriano. 1999. Pseudo-outbreak of Aeromonas hydrophila isolates related to endoscopy. *Journal of Hospital Infection.* 41:313-316.

Farmer, J.J. III, M.J. Arduino, and F.W. Hickman-Benner. 1992. The genera Aeromonas and Plesiomonas. In Balows. A., H.G. Truper, M. Dworkin, W. Harder, and K.H. Schleifer (Eds.). The Prokaryotes. 2nd Ed. Springer, New York. 2938-2961.

Falkinham J. O. III. 1996. Epidemiology of infection by nontuberculous mycobacteria. *Clinical Microbiology Reviews.* 9:177-215.

Falkinham J. O. III, C. D. Norton, and M. W. LeChevallier. 2001. Factors influencing numbers of Mycobacterium avium, Mycobacterium intracellulare, and other mycobacteria in drinking water distribution systems. *Applied and Environmental Microbiology*. 67(3):1225-1231.

Falkinham, J. O. III. 2002. Nontuberculous mycobacteria in the environment. *Clinics in Chest Medicine.* 23(3):529-551.

Fournier, S., O. Liguory, V. Garrait, J.P. Gagneux, C. Sarfati, F. Derouin, and J.M. Molina. 1998. Microsporidiosis due to Enterocytozoon bieneusi infection as a possible cause of traveller's diarrhea. *European Journal of Clinical Microbiology and Infectious Diseases.* 17:743-744.

Fournier, S.O., Liguory, M. Santillana-Hayat, E. Guillot, C. Sarfati, N. Dumoutier, J.M. Molina, and F. Derouin. 2000. Detection of microsporidia in surface water: a one-year follow-up study. *FEMS Immunology and Medical Microbiology*. 29:95-100.

Foy, H.M. 1997. Adenoviruses. In: Viral Infections of Humans. A.S. Evans and R.A. Kaslow, Eds. Fourth edition. Plenum Medical Book Company, New York and London. pp. 119-138.

Franzen, C. and A. Müeller. 1999. Cryptosporidia and microsporidia- waterborne diseases in the immunocompromised host. *Diagnostic Microbiology and Infectious Disease*. 34:245-262.

Gasbarrini, A., E. Carloni, G. Gasbarrini, and A. Menard, A. 2003. Helicobacter pylori extragastric diseases- Other Helicobacters. *Helicobacter.* 8(suppl.1):68-76.

Gavriel, A.A., J.P.B. Landre, and A.J. Lamb. 1998. Incidence of mesophilic Aeromonas within a public drinking water supply in north-east Scotland. *Journal of Applied Bacteriology*. 84:383-392.

Gerba, C.P., N. Nwachuku, and R.K. Riley. 2003a. Disinfection resistance of waterborne pathogens on the United States Environmental Protection Agency's Contaminant Candidate List (CCL). *Journal of Water Supply Research and Technology*. 52:81-94.

Gerba, C.P., K.R. Riley, N. Nwachuku, H. Ryu, and M. Abbaszadegan. 2003b. Removal of Encephalitozoon intestinalis, calicivirus, and coliphages by conventional drinking water treatment. Journal of Environmental Science and Health - Part A: Toxic/Hazardous Substances and Environmental Engineering. 38(7):1259-1268.

Griffin, D.W., C.J. Gibson III, E.K. Lipp, K. Riley, J.H. Paul III, and J.B. Rose. 1999. Detection of viral pathogens by reverse transcriptase PCR and of microbial indicators by standard methods in the canals of the Florida Keys. *Applied and Environmental Microbiology*. 65:4118-4125.

Hegarty, J.P., M.T. Dowd, and K.H. Baker. 1999a. Occurrence of Helicobacter pylori in surface water in the United States. *Journal of Applied Microbiology*. 87:697-701.

Hegarty, J.P., Y.L. Ma, C.M. Lebo, T.L. Konopka, and K.H. Baker. 1999b. Water-borne transmission of Helicobacter pylori: Convergence of environmental and clinical observations. Paper presented at; 99th General Meeting, American Society for Microbiology; 1999b June 2; Chicago, IL.

Hejkal, T.W., F.M. Wellings, A.L. Lewis, and P.A. LaRock. 1981. Distribution of viruses associated with particles in wastewater. *Applied and Environmental Microbiology*. 41:628-634.

Hejkal, T.W., B.H. Keswick, R.L. LaBelle, C.P. Gerba, Y. Sanchez, G. Dreesman, B.A. Hafkin, and J.L. Melnick. 1982. Viruses in a community water supply associated with an outbreak of gasteroenteritis and infectious hepatitis. *Journal of the American Water Works Association*. 74:731-733.

Hester, J.D., H.D.A. Lindquist, A.M. Bobst, and F.W. Schaeffer, III. 2000. Fluorescent In Situ Detection of Encephalitozoon hellem Spores with a 6-Carboxyfluorescein-Labeled Ribosomal RNA-Targeted Oligonucleotide Probe. *Journal of Eukaryotic Microbiology*. 47:299-308.

Hierholzer, J.C. 1992. Adenovirus in the immunocompromised host. *Clinical Microbiology Reviews.* 5:262-274.

Hitzfeld, B. C., S. J. Hoger, and D. R. Dietrich. 2000. Cyanobacterial toxins: removal during drinking water treatment, and human risk assessment. *Environmental Health Persp*ectives. 1:113-122.

Holst, T., N.O. Jorgensen, C. Jorgensen, and A. Johansen. 2003. Degradation of microcystin in sediments at oxic and anoxic, denitrifying conditions. *Water Research*. 37(19):4748-4760.

Horne, A. J. and C.R. Goldman. 1994. Limnology. San Francisco: McGraw-Hill.

Horsburgh, C. R., Jr. 1991. Mycobacterium avium complex infection in the acquired immunodeficiency syndrome. *New England Journal of Medicine*. 324(19):1332-1338.

Horsburgh, C. R., Jr., B. G. Metchock, J. E. McGowan, Jr., and S. E. Thompson. 1992. Clinical implications of recovery of Mycobacterium avium complex from the stool or respiratory tract of HIV-infected individuals. *AIDS.* 6(5):512-514.

HPLC Users Group. 1999. Mycolic acid pattern standards for HPLC identification of mycobacteria. CDC, Atlanta, GA.

Hulten, K., H. Enroth, T. Nystrom, and L. Engstrand. 1998. Presence of Helicobacter species DNA in Swedish water. *Journal of Applied Microbiology*. 85: 282-262.

Hurst, C.J. 1991. Presence of enteric viruses in freshwater and their removal by the conventional drinking water treatment process. *Bulletin of the World Health Organization*. 69:113-119.

Hurst, C.J., W.H. Benton, and K.A. McClellan. 1988. Suppression of viral replication by guanidine: a comparison of human adenoviruses and enteroviruses. *Journal of Virological Methods.* 22:1-11.

Hutin, Y.J.F., M.N. Sombardier, O. Ligoury, C. Sarfati, F. Derouin, J. Modai, and J.M. Molina. 1998. Risk factors for intestinal microsporidiosis in patients with human immunodeficiency virus infection: A case control study. *Journal of Infectious Diseases*. 178:904-907.

International Association for Research on Cancer (IARC). 1994. Infection with Helicobacter pylori (Group 1): 5. Summary of Data Reported and Evaluation. Vol. 61, pp. 177. Available on the Internet at: http://monographs.iarc.fr/htdocs/monographs/vol61/m61-3.htm. Accessed May 3, 2005. Last updated August 26, 1997.

Iivanainen, E., P. J. Martikainen, P. Vaananen, and M. L. Katila. 1999. Environmental factors affecting the occurrence of mycobacteria in brook sediments. *Journal of Applied Microbiology*. 86(4):673-681.

Inderlied, C. B., C. A. Kemper, and L. E. Bermudez. 1993. The Mycobacterium avium complex. *Clinical Microbiology Reviews*. 6:266-310.

Isakov, V and P. Malfertheiner. 2003. Helicobacter pylori and nonmalignant diseases. *Helicobacter*. 8(supp 1):36-43.

Janda, J.M. and S.L. Abbott. 1998. Evolving concepts regarding the genus Aeromonas: an expanding panorama of species, disease presentations, and unanswered questions. *Clinical Infectious Diseases.* 27:332-344.

Jiang, S., R. Noble, and W. Chu. 2001. Human adenoviruses and coliphages in urban runoff-impacted coastal waters of southern California. *Applied and Environmental Microbiology*. 67(1):179-184.

Jiang, X., D.Y. Graham, K.N. Wang, and M.K. Estes. 1990. Norwalk virus genome cloning and characterization. *Science.* 250:1580-1583.

Jochimsen, E.M., W.W. Carmichael, J.S. An, D.M. Cardo, S.T. Cookson, C.E. Holmes, M.B. Antunes, D.A. de Melo Filho, T.M. Lyra, V.S. Barreto, S.M. Azevedo, and W.R. Jarvis. 1998. Liver failure and death after exposure to microcystins at a hemodialysis center in Brazil. *New England Journal of Medicine.* 338: 873-878.

Johnson, C.H., E.W. Rice, and D.J. Reasoner. 1997. Inactivation of Helicobacter pylori by chlorination. *Applied and Environmental Microbiology*. 63:4969-4970.

Kahana, L. M. and J. M. Kay. 1997. Pneumonitis due to Mycobacterium avium complex in hot tub water: infection or hypersensitivity? *Chest.* 112(6):1713-1714.

Kaplan, J.E., R.A. Goodman, L.B. Schonberger, E.C. Lippey, and G.W. Gary. 1982. Gasteroenteritis due to a Norwalk virus: An outbreak associated with a municipal water system. *Journal of Infectious Diseases*. 146(2):190- 197.

Karner, D.A, J.H. Standridge, G.W. Harrington, and R.P. Barnum. 2001. Microcystin algal toxins. *Journal of the American Water Works Association.* 93(8): 72-81.

Kotler, D.P. and J.M. Orenstein. 1999. Clinical syndromes associated with microsporidiosis. In: The Microsporidia and Microsporidiosis. M. Wittner and L.M. Weiss, Eds. ASM Press, Washington, D.C.

Kramer, J.P. 1970. Longevity of microsporidian spores with special reference to Octosporea muscae-domesticae Flu. *Acta Protozoologica*. 8:217-224.

Krikelis, V., N. Spyrou, P. Markoulatos, and C. Serie. 1985. Seasonal distribution of enteroviruses and adenoviruses in domestic sewage. *Canadian Journal of Microbiology*. 31(1):24-25.

Lanciers, S., B. Hauser, Y. Vandenplas, and U. Blecker. 1996. The prevalence of Helicobacter pylori positivity in asymptomatic children of different ethnic backgrounds living in the same country. *Ethnicity and Health.* 1:169-173.

Lau, S. M., M. Y. Peng, and F. Y. Chang. 2000. Outcomes of Aeromonas bacteremia in patients with different types of underlying disease. *Journal of Microbiology, Immunology and Infection*. 33(4):241-247.

LeBaron, C.W., N.P. Furutan, J.F. Lew, J.R. Allen, V. Gouvea, C. Moe, and S.S. Monro. 1990. Viral agents of gasteroenteritis. Public health importance and outbreak management. *Morbidity and Mortality Weekly Report.* 39:1-24.

Le Dantec, C., J-P Duguet, A. Montiel, N. Dumoutier, S. Dubrou, and V. Vincent. 2002. Occurrence of Mycobacteria in Water Treatment Lines and in Water Distribution Systems. *Applied and Environmental Microbiology*. 68(11): 5318–5325.

Lodder, W.J., J. Vinjéé, R. van de Heide, A.M. de Roda Husman, E.J.T.M. Leenen, and M.P.G. Koopmans. 1999. Molecular detection of norwalk-like caliciviruses in sewage. *Applied and Environmental Microbiology.* 65(12):5624-5627.

Loge, F.J., D.E. Thompson, and D.R. Call. 2002. PCR detection of specific pathogens in water: a risk-based analysis. *Environmental Science and Technology*. 36(12): 2754-2759.

Maddox, J.V. 1973. The persistence of the microsporidia in the environment. Miscellaneous *Publications of the Entomological Society of America*. 9:99-106.

Madigan, M.T., J.M. Martinko, and J. Parker. 1997. Brock Biology of the Microorganisms. Upper Saddle River, New Jersey, Prentice Hall.

Malaty, H.M., D.Y. Graham, P.D. Klein, D.G. Evans, E. Adam, and D.J. Evans. 1991. Transmission of Helicobacter pylori infection. Studies in families of healthy individuals. *Scandinavian Journal of Gastroenterology*. 26: 927-32.

Martino, T.A., P. Liu, M. Petric, and M.J. Sole. 1995. Enteromyocarditis and dilated cardiomyopathy: A review of clinical and experimental studies. In: Human Enterovirus Infections. H.A. Rothbart, Ed. ASM Press, Washington, D.C.: pp. 291-351.

Matsubayashi, H., T. Koike, I. Mikata, H. Takei, and S. Hagiwara. 1959. A case of encephalitozoon-like body in man. *Archives of Pathology*. 67:181-187.

McKeown, I., P. Orr, S. Macdonald, A. Kabani, R. Brown, G. Coghlan, M. Dawood, J. Emil, M. Sargent, G. Smart, and C.N. Bernstein. 1999. Helicobacter pylori in the Canadian arctic: seroprevalence and detection in community water samples. *American Journal of Gastroenterology*. 94:1823-1829.

Melnick, J.L. 1997. Poliovirus and other enteroviruses. In: Viral Infections of Humans. A.S. Evans and R.A. Kaslow, Eds. Fourth edition. Plenum Medical Book Company, New York and London. pp. 583-663.

Menotti, J., B. Cassinat, C. Sarfati, O. Liguory, F. Derouin, and J.M. Molina. 2003. Development of a real-time PCR assay for quantitative detection of Encephalitozoon intestinalis DNA. *Journal of Clinical Microbiology*. 41(4):1410-1413.

Minnikin, D.E. 1991. Chemical principles in the organization of lipid components in the mycobacterial cell envelope. *Research in Microbiology*. 142:423-427.

Minor, P. 1998. Picornaviruses. In: Virology. B.W.J. Mahy and L. Collier, Eds. Edward Arnold London: pp. 485-509.

Modlin, J.F. 1986. Perinatal echovirus infection: Insights from a literature review of 61 cases of serious infection and 16 outbreaks in nurseries. *Reviews of Infectious Diseases*. 8:918-926.

Morbidity and Mortality Weekly Report (MMWR). 1990. Viral agents of gasteroenteritis. Public health importance and outbreak management. Publication of the Centers for Disease Control and Prevention. April 27, 1990. 39(RR-5):1-24.

Moreno, Y., M.A. Ferrus, J.L. Alonso, A. Jimenez, and J. Hernandez. 2003. Use of fluorescent in situ hybridization to evidence the presence of Helicobacter pylori in Water. *Water Research.* 37(9):2251-2256.

Moyer, N.P. 1987. Clinical significance of Aeromonas species isolated from patients with diarrhea. *Journal of Clinical Microbiology*. 25: 2044-2048.

Naumovitz, D.W., M.M. Marshall, K.F. Grahn, T.P. Clancy, and C.R. Sterling. 1998. Treatment of Encephalitozoon sp. with ozone or pulsed UV light. Water Quality Technology Conference Proceedings, San Diego, CA.

Nichols, G.L. 1996. Fact Sheets on Emerging Waterborne Pathogens: Final Report to the Department of the Environment: Aeromonas hydrophila. WRc and Public Health Laboratory Service. DWI 4248/1.

Nikaido, H., S.H. Kim, and E.Y Rosenberg. 1993. Physical organization of lipids in the cell wall of Mycobacterium chelonae. *Molecular Microbiology*. 8:1025-1030.

Norton, C., M. LeChevallier, J.O. Falkinham, M. Williams, and R. Taylor. 1999. Mycobacterium avium in drinking water. Presented at AWWA International Symposium on Waterborne Pathogens, August 29-September 1, 1999, Milwaukee, WI.

Notkins, A.L. 1977. Virus-induced diabetes mellitus. *Archives of Virology*. 54:1-17.

Orenstein, J.M, D.T. Dieterich, and D.P. Kotler. 1992. Systemic dissemination by a newly recognized intestinal microsporidia species in AIDS. *AIDS*. 6:1143-1150.

Orenstein, J.M., E.S. Didier, R.B. Mertens, S.S. Frankel, A.T. Yachnis, and H.P. Gaetz. 1997. Disseminated microsporidiosis in AIDS: are any organs spared? *AIDS.* 11:385-386.

Ozbek, A. and O. Aktas. 2003. Identification of three strains of Mycobacterium species isolated from clinical samples using fatty acid methyl ester profiling. *Journal of International Medical Research.* 31(2):133-140.

Papapetropoulou, M. and A.C. Vantarakis. 1998. Detection of Adenovirus outbreak at a municipal swimming pool by nested PCR amplification. *Journal of Infection*. 36:101-103.

Parshionikar, S.U., S. Willian-True, G.S. Fout, D.E. Robbins, S.A. Seys, J.D. Cassady, and R. Harris. 2003. Waterborne outbreak of gasteroenteritis associated with a norovirus. *Applied and Environmental Microbiology*. 69(9):5263- 5268.

Pauschnger, M., N.E. Bowles, F.J. Fuentes-Gaarcia, V. Pham, U.Küühl, P.L. Schwimmbeck, H.-P. Schultheiss, and J.A. Towbin. 1999. Detection of adenoviral genome in the mycocardium of adult patients with idiopathic left ventricular dysfunction. *Circulation*. 99:1348-1354.

Payment P., M. Tremblay, and M. Trudel. 1985. Relative resistance to chlorine of poliovirus and coxsackievirus isolates from environmental sources and drinking water. *Applied and Environmental Microbiology.* 49(4):981- 983.

Peng, X., J. Zhang, S. Wang, Z. Lin, and W. Zhang. 2002. Immuno-capture PCR for detection of Aeromonas hydrophila. *Journal of Microbiological Methods*. 49(3):335-338.

Pettibone, G.W. 1998. Population dynamics of Aeromonas spp. in an urban river watershed. *Journal of Applied Microbiology*. 85:723-730.

Pilotto, L.S., E.V. Kliewer, R.D. Davies, M.D. Burch, and R.G. Attewell. 1999. Cyanobacterial (blue-green algae) contamination in drinking water and perinatal outcomes. *Australian and New Zealand Journal of Public Health*. 23:154-158.

Pina, S., M. Puig, F. Lucerna, J. Jofre, and R. Girones. 1998. Viral pollution in the environment and in shellfish: human adenovirus detection by PCR as an index of human viruses. *Applied and Environmental Microbiology*. 64(9): 3376-3382.

Pouria, S., A. de Andrade, J. Barbosa, R.L. Cavalcanti, V.T. Barreto, C.J. Ward, W. Preiser, G.K. Poon, G.H. Neild, and G.A. Codd. 1998. Fatal microscytin intoxication in haemodialysis unit in Caruaru, Brazil. *Lancet.* 352:21-26.

Powell, K.L., R.G. Taylor, A.A. Cronin, M.H. Barrett, S. Pedley, J. Sellwood, S.A. Trowsdale, and D.N. Lerner. 2003. Microbial contamination of two urban sandstone aquifers in the UK. *Water Research*. 37(2):339- 352.

Reynolds, K.A., C.P. Gerba, M. Abbaszadegan, and I.L. Pepper. 2001. ICC/PCR detection of enteroviruses and hepatitis A virus in environmental samples. *Canadian Journal of Microbiology*. 47(2):153-157.

Rinder, H., S. Katzwinkel-Wladarsch, and T. Loscher. 1997. Evidence for the existence of genetically distinct strains of Enterocytozoon bieneusi. *Parasitology Research.* 83:670-672.

Romero C., A. Hamdi, J.F. Valentine, and S.A. Naser. 2005. Evaluation of surgical tissue from patients with Crohn's disease for the presence of Mycobacterium avium subspecies paratubuerculosis DNA by in situ hybridization and nested polymerase chain reaction. *Inflammatory Bowel Diseases*. 11(2):116-25.

Rowe, W.P., R.J. Huebner, L.K. Gilmore, R.H. Parrott, and T.G. Ward. 1953. Isolation of a cytopathogenic agent from human adenoids undergoing spontaneous degeneration in tissue culture. *Proceedings of the Society for Experimental Biology and Medicine.* 84:570-573.

Rowland, M. and B. *Drumm. 1998. Clinical significance of Helicobacter infection in children.* British Medical Bulletin. 54: 95-103.

Sasaki K, Y. Tajui, M. Sata, Y. Fuji, F. Matsubara, M. Zhao, S. Shimizu, A. Toyonaga, and K. Tanikawa. 1999. Helicobacter pylori in the Natural Environment. *Scandinavian Journal of Infectious Diseases.* 31: 275-279.

Shin, G.-A., D. Battigelli, and M.D. Sobsey. 1998. Reduction of Norwalk virus, poliovirus1, and colophage MS2 by free chlorine, chlorine dioxide, and ozone disinfection of water. Water Quality Technology Conference November 1-4, 1998, San Diego, CA.

Smith, H.R. and T. Cheasty. 1998. Diarrhoeal Disease Due to Escherichia coli and Aeromonas. In W.J. Hausler & M. Sussman (Eds.), Microbiology and Microbial Infections. (pp. 513-529). New York: Oxford University Press.

Sobottka, I., H. Albrecht, J. Schottelius, C. Schmetz, M. Bentfeld, R. Laufs, and D.A. Schwart. 1995. Self-limited traveler's diarrhea to a dual infection with Enterocytozoon bieneusi and Cryptosporidium parvum in an immunocompetetent HIV-negative child. *European Journal of Clinical Microbiology and Infectious Diseases*. 14(10):919-920.

Sobsey, M. D., T. Fuji, and P. A. Shields. 1988. Inactivation of hepatitis A virus and model viruses in water by free chlorine and monochloramine. *Water Science and Technology*. 20:385-391.

Sorel, N., E. Guillot, M. Thellier, I. Accoceberry, A. Datry, L. Mesnard-Rouiller, and M. Miegeville. 2003. Development of an immunomagnetic separation-polymerase chain reaction (IMS-PCR) assay specific for Enterocytozoon bieneusi in water samples. *Journal of Applied Microbiology*. 94(2):273-279.

Sparfel, J.M., C. Sarfati, O. Ligoury, B. Caroff, N. Dumontier, B. Gueglio, E. Billaud, F. Raffi, J.M. Molina, M. Miegeville, and F. Derouin. 1997. Detection of microsporidia and identification of Enterocytozoon bieneusi in surface water by filtration followed by specific PCR. *Journal of Eukaryotic Microbiology*. 44:78S.

Staat, M.A., D. Kruszon-Moran, G.M. McQuillan, and R.A. Kaslow. 1996. A population-based serologic survey of Helicobacter pylori infection in children and adolescents in the United States. *Journal of Infectious Diseases*. 174:1120-1123.

Swanson, D. S., X. Pan, M. W. Kline, R. E. McKinney, Jr., R. Yogev, L. L. Lewis, M. T. Brady, G. D. McSherry, W. M. Dankner, and J. M. Musser. 1998. Genetic diversity among Mycobacterium avium complex strains recovered from children with and without human immunodeficiency virus infection. *Journal of Infectious Diseases*. 178(3):776-782.

Tani, N., N. Spyrou, P. Markoulatos, and C. Serie. 1995. Seasonal distribution of adenoviruses, enteroviruses and reoviruses in urban river water. *Microbiology and Immunology*. 39(8):577-580.

Taylor, R.H., J.O. Falkinham III, C.D. Norton, and M.W. LeChevallier. 2000. Chlorine, chloramine, chlorine dioxide, and ozone susceptibility of Mycobacterium avium. *Applied and Environmental Microbiology*. 66(4):1702-1705.

Thorel, M.F., M.C. Blom-Potar, and N. Rastogi. 1990a. Characterization of Mycobacterium paratuberculosis and "wood-pigeon" mycobacteria by isoenzyme profile and selective staining of immunoprecipitates. *Research in Microbiology*. 141(5):551-561.

Thorel, M.F., M. Krichevsky, and V.V. Levy-Frebault. 1990b. Numerical taxonomy of mycobactin-dependent mycobacteria, emended description of Mycobacterium avium, and description of Mycobacterium avium subsp. avium subsp. nov., Mycobacterium avium subsp. paratuberculosis subsp. nov., and Mycobacterium avium subsp. silvaticum subsp. nov. *International Journal of Systematic Bacteriology*. 40(3):254-260.

Thurston, J.A., S.E. Dowd, P.M. Watt, R. Enriquez, F.D. Vladich, P. Moroyoqui, I.L. Pepper, and C.P. Gerba. 1999. Occurrence of microsporidia in waters used for irrigation in Latin America and the United States. Abstract, American Society for Microbiology 99th General Meeting, Chicago, Illinois, American Society for Microbiology.

Tomar, B. S. 2001. Intestinal infections. *Indian Journal of Pediatrics*. 68(Ss-3):S8-S19.

Turner, M., G.R. Istre, H. Beauchamp, M. Baum, and S. Arnold. 1987. Community outbreak of adenovirus type 7a infections associated with a swimming pool. *Southern Medical Journal*. 80:712–715.

Undeen, A.H., M.A. Johnson, and J.J. Becnel. 1993. The effects of temperature on the survival of Edhazardia aedis (Microspora, Ambylosporidae), a pathogen of Aedes aegypti. *Journal of Invertebrate Pathology.* 61:303-307.

United States Environmental Protection Agency (USEPA). 1999. Mycobacteria: Health Advisory. EPA Report 822-B-01-007. Available on the Internet at: http://www.epa.gov/waterscience/criteria/humanhealth/microbial/mycobacteriaha.pdf.

USEPA. 2000. Draft Method 1605: Aeromonas in Finished Water by Membrane Filtration. EPA Report 821-R-00-026.

USEPA. 2001. Creating a Cyanotoxin Target List for the Unregulated Contaminant Monitoring Rule. U.S. EPA meeting on cyanobacteria and their toxins. Cincinnati, OH.

USEPA. 2002. Treatability screening for CCL microbial contaminants. EPA Report 822-R-01-002.

van Doorn, L.J., Y. Henskens, and N. Nouhan. 2000. The efficacy of laboratory diagnosis of Helicobacter pylori infections in gastric biopsy specimens is related to bacterial density and vacA, CagA, and iceA genotypes. *Journal of Clinical Microbiology*. 38:13-17.

van Soolingen, D. 2001. Molecular epidemiology of tuberculosis and other mycobacterial infections: main methodologies and achievements. *Journal of Internal Medicine.* 249:1-26.

Vassili, I and P. Malfertheiner. 2003. Helicobacter pylori and nonmalignant diseases. *Helicobacter*. 8(suppl. 1):36-43.

Vila, J., J. Ruiz, F. Gallardo, M. Vargas, L. Soler, M. J. Figueras, and J. Gascon. 2003. Aeromonas spp. and traveler's diarrhea: clinical features and antimicrobial resistance. *Emerging Infectious Diseases.* 9(5):552-555.

Vivier, J.C, C.G. Clay, and W.O.K. Grabow. 2001. Detection and rapid differentiation of human enteroviruses in water sources by restriction enzyme analysis. *Water Science and Technology*. 43(12):209-212.

Vivier, J.C., M.M. Ehlers, and W.O.K. Grabow. 2004. Detection of enteroviruses in treated drinking water. *Water Research*. 38:2699-2705.

von Reyn, C.F., T.W. Barber, R D. Arbeit, C.H. Sox, G.T. O'Connor, R.J. Brindle, C.F. Gilks, K. Hakkarainen, A. Ranki, and C. Bartholomew. 1993. Evidence of previous infection with Mycobacterium avium-Mycobacterium intracellulare complex among healthy subjects: an international study of dominant mycobacterial skin test reactions. *Journal of Infectious Diseases*. 168(6):1553-1558.

von Reyn, C.F., J.N. Maslow, T.W. Barber, J.O. Falkinham, 3rd, and R.D. Arbeit. 1994. Persistent colonisation of potable water as a source of Mycobacterium avium infection in AIDS. *Lancet*. 343(8906):1137-1141.

Wang, G., C.G. Clark, C. Liu, C. Pucknell, C.K. Munro, T.M. Kruk, R. Caldeira, D.L. Woodward, and F.G. Rodgers. 2003. Detection and characterization of the hemolysin genes in Aeromonas hydrophila and Aeromonas sobria by multiplex PCR. *Journal of Clinical Microbiology*. 41(3):1048-1054.

Weiss, L.M. and E.M. Keohane. 1997. The uncommon gastrointestinal protozoa: Microsporidia, Blastocystis, Isospora, Dientamaoeba, and Balantidium. *Current Clinical Topics in Infectious Diseases*. 17:147-187.

Wittner, M. 1999. Historic perspective on the microsporidia: Expanding Horizons. In: The Microsporidia and Microsporidiosis. M. Wittner and L.M. Weiss, Eds. ASM Press, Washington, D.C.

Wolinsky, E. 1995. Mycobacterial lymphadenitis in children: a prospective study of 105 nontuberculous cases with long-term follow-up. *Clinical Infectious Diseases*. 20(4):954-963.

Wolk, D.M., C.H. Johnson, E.W. Rice, M.M. Marshall, K.F. Grahn, C.B. Plummer, and C.R. Sterling. 2000. A spore counting method and cell culture model for chlorine disinfection studies of Encephalitozoon syn. Septata intestinalis. *Applied and Environmental Microbiology*. 66(4):1266-1273.

World Health Organization (WHO). 1998. Guidelines for Safe recreational-water environments: coastal and fresh-waters. Geneva.

WHO. 2004. Guidelines for Drinking-water Quality, Third Edition. Volume 1: Recommendations. Geneva. Available on the Internet at: http://www.who.int/water_sanitation_health/dwq/gdwq3/en/. Accessed July 26, 2005.

Yoo, R.S., W.W. Carmichael, R.C. Hoehn, and S.E. Hrudey. 1995. Cyanobacterial (Blue-Green Algal) Toxins: A Resource Guide. Denver: American Water Works Association Research Foundation.

Zuberbier, T. 2003. Urticaria. *Allergy*. 58:1224-1234.